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THE DIAGNOSIS AND TREATMENT OF SUBDURAL HEMATOMATA*

A Report of Sixty-two Cases

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IN a paper written thirty-one years ago on subdural hematomata under the name of Pachymeningitis Haemorrhagica, my father[1] stated that the interest up to that time (in this disease) had been almost exclusively pathological and that he wished to emphasize that it should be more frequently surgical. I believe that in essence this statement is as true today as it was then. Because of our present failure to recognize the acute stages of so-called chronic subdural hematoma except at postmortem, we fail to identify the latter as a late stage of the former, and neglect the majority in favor of the minority. This paper presents my evidence in support of these statements and summarizes data obtained from a study of 62 subdural hemorrhages admitted to the Boston City Hospital during four and one-half years. Both "acute" and "chronic" forms are included. Forty-one were on the Neurosurgical Service under my care. Of the remaining 21, 10 occurred on the medical services and the rest (11) on the general surgical side.

LITERATURE. A survey of the literature for the past 10 years demonstrates that the clinical or surgical side of this problem has been largely investigated through information derived only from the study of small groups of cases. Seventy-three articles published during this period have been available for review. Of these thirty-seven were single case reports. In fifty-four the authors' experience was limited to no more than five and in sixty-six to no more than 10 cases. Five offered no more proof than the author's opinion that he was dealing with the disease in question. Of the remaining two, one by Jelsma[16] is a compilation of forty-two cases selected from the literature to which he has added two of his own. The other records Coleman's[11] personal experience with twenty instances and includes both the "acute" and "chronic" forms of the disease. Though not given in such detail I should infer that his conclusions are fundamentally similar to mine and certainly from the surgical point of view are the only ones that merit serious attention from among this group.

Isolated observations of value when taken in conjunction with the whole mass of data were frequent however. For example: it has thus been demonstrated that subdural hematoma may occur one hour, five and a half and sixteen months postpartum[17, 18, 5], that it may remain quiescent long enough to permit complete calcification even at the early age of eleven years[19, 20], that it may be associated with syphilis[21], scurvy[5] or cerebral tumor[22, 11], that it is uncommon in congenital syphilities under two years of age[23], that children are not immune[24, 25, 26, 5, 7] and that it is a rare but possible occurrence in the posterior fossa[27]. Encephalography and vetriculography have both been successfully used as diagnostic measures. Removal of the clot has been successfully carried out through trephine holes[14], by bone flaps[12], and by means of multiple operations[28], and finally a therapeutic bone defect in the skull has been closed by a platinum plate[29] in the course of the treatment of this disease.

INCIDENCE. Autopsy statistics from Chicago by Le Count and Apfelbach[2] and from New York by Vance[3] list subdural hematomata as among the commoner complications of cranio-cerebral injuries. (Chart I.). The former found 300 among 374 fractures of the skull and the latter recognized it as the cause of death in 132 out of 512 autopsies, or twenty-five per cent. At the Boston City Hospital for a period of four years up to 1933 it has been noted thirty-six times among 1352 head injuries. This is at the rate of 2.6 per cent. These cases were all on the general surgical or medical services. With one exception the diagnoses were either not confirmed or were made at autopsy. Conversely in the past twelve months from January, 1933 on the Neurosurgical Service at the same institution thirty-five of the thirty-seven proved subdural hemorrhages among 201 head injuries were diagnosed clinically. This is at the rate of 17 per cent. It is obvious that previous to 1933 at the Boston City Hospital, many such cases have not

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only been undiagnosed but their presence has not even been suspected. Once the frequency of this condition had been appreciated and diagnostic methods improved to a point where early treatment could be instituted, many more cases were recognized. The resultant drop in mortality among verified cases from 100 per cent on the general services to 41 per cent on the Neurosurgical Service has been striking.

CHART I

INCIDENCE

Time Involved	Total Cases Injury	Subdural Hematomata		Method of Diagnosis Clinical Verified	Clinical Not Verified	Post-mortem
Four years to 1933—Whole hospital	1352	36	2.6%	1	10	25
Jan., 1933, to Jan., 1934—One year Neurosurgical Service only	201	37	17.0%	35	0	2
Vance's figures	512	132	25.0%	—	—	132

ETIOLOGY. Trauma is generally recognized as the chief and by some authors as the only cause of this condition. Henschen[4] however points out that there may be other causes, mentioning among other things, disease of the blood and blood-forming structures, arteriosclerotic changes, syphilis and even experimental avitaminosis. (Chart II.) Nutritional diseases in children are also a frequent cause[5]. Four of my cases were complicated in this way, three by syphilis and one by the presence of hemophilia.

The source of the hemorrhage in the "chronic" form is stated to be for the most part either from a rupture of a bridging cortical vein or of a tributary to one of the large venous sinuses. Although not necessarily so, it is probable that this is true of most of the cases in whom symptoms develop slowly. Taken in relation to the whole group, however, this is only a small number. Both Le Count[2] and Vance[3] find these to be unusual or rare origins for subdural hemorrhage in autopsied cases. My figures are in agreement with theirs in that fractures of the skull, and laceration and contusion of the brain are much more frequently the causative agents of the bleeding than the rupture of a single vein. The hemorrhage may also be so small as to escape notice even after the subdural space is open. In such cases the symptoms are caused by an associated collection of fluid. Naffziger[6] directed attention to this condition in 1924 and more recently (1932) Peet and Kahn[7] described it in infants. It was noted seven times among my cases.

PATHOLOGY. A detailed study of the histopathology of the subdural hematomata removed at operation, particularly in regard to their microscopic structure and its relationship to the time elapsed between their formation and removal must be reserved for later presentation. Suffice it to say that certainly within three hours after receipt of the injury and the presumed formation of the clot, a single layer of fibroblastic cells has already extended from the dura well onto the surface of the former. The process that eventually is to terminate by encapsulation of the free blood with organization of that portion adjacent to the capsule plus associated liquefaction of the more distant central parts has begun.

CHART II

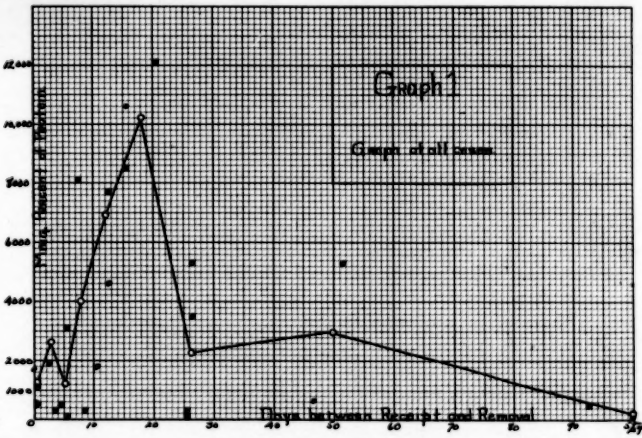
SOURCE OF BLEEDING

	Bilateral	Ruptured Vein	Lacerated or Contused Brain	Fractured Skull	Subdural Fluid	No Note
Source	11	3	24	15	7	22

	Pneumonia and Empyema	Cerebral and Cerebellar	Hemophilia	Meningitis	Known Alcoholic	Syphilis
Complications	1	1	1	1	23	3

There is abundant indirect clinical evidence, based upon the frequent occurrence of a "latent period" in the development of symptoms, that such clots may slowly increase in size. The favorite explanation of this phenomenon has been to predicate repeated or slow seeping hemorrhages. These occur inside the clot from thin-walled blood spaces or "giant capillaries" and may produce multiple neo-membranes[30]. Such a condition is usually fatal however and is not a necessary prerequisite for a latent period. An equally reasonable explanation of this phenomenon has been offered recently by Gardner[8] and also during the past year, although apparently independently by Fischer and de Morsier[31]. The former has shown experimentally that a given subdural clot which presents only a mini-

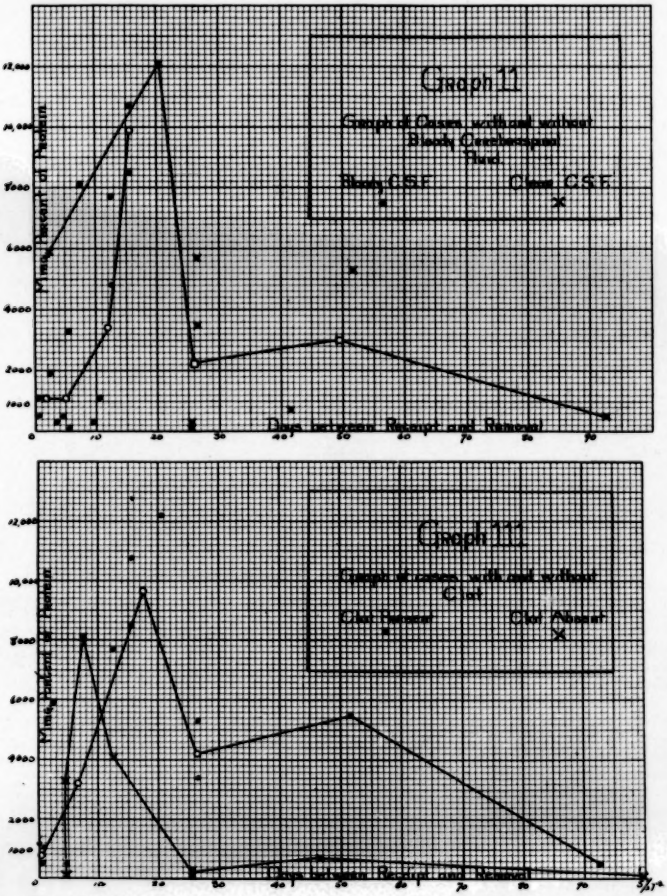
mum of signs at the time of its original formation will, after partial liquefaction, absorb by dialysis from the subarachnoid space sufficient fluid to enlarge it from 39 to 103 per cent. This flow results from the proximity of a highly concentrated protein solution to another solution in which the protein concentration is very much lower, the sole means of separating the two being the arachnoid which acts as a dialyzing membrane. I have been able to show that this experimental relationship holds true also in the human being. My figures when plotted graphically (graph 1) further suggest that there is a



period during which the protein concentration of the fluid part of the clot increases. This is due in part to liquefaction of the clot. In addition the protein subsequently breaks down into smaller molecules*. Both these factors produce a rising osmotic pressure within the solution. A peak is then reached and thereafter there is a steady dilution with resultant lowering of the concentration, although at no time up to five and a half years does the subdural protein ever descend to the level of that in the subarachnoid space. This dilution proceeds rapidly by osmosis until blood serum concentration levels are reached. After that the process is slowed up, the rate depending on the proximity of the fluid to a capillary bed with resultant provision for the diffusion therein of the end products of the protein destruction. Based on a study of 25 fluids removed at operation and analyzed for me in F. Fremont-Smith's laboratory I have found that during the first twenty-four hours after injury the protein content averages 1100 mg. per cent, the low being 500 and the high 1700. An average peak of 10,200 mg. per cent is reached between the fifteenth and twenty-first days. The highest concentration occurred on the twenty-first day and was 12,168 mg. per cent which is almost double that found in blood serum. Thereafter the fall is rapid to the twenty-seventh day, the average content at this time being 2200 mg. per cent. From this point to the ninetieth day the fall is much slower with a shrinkage of only 700 mg. per cent. Even at five and a half years the protein concentration in one case was still 140 mg. per cent. On these figures, which are admittedly too few to be finally conclusive in detail, it seems to me fair to assume that there is a liquefaction and breakdown of the clot extending from the first to between the fifteenth and twenty-first days. This solution then becomes more and more diluted; rapidly for the first ten days and progressively more slowly thereafter, without ever actually reaching the low concentration level of the cerebrospinal fluid even after five and a half years. The corollary to this process of dilution is necessarily an increase in the amount of the diluted fluid and therefore an increase in the size of the hematoma. This is expressed clinically in terms of symptoms of an expanding lesion and occurs without necessarily any further addition in the form of secondary or late hemorrhages. Blood in the cerebrospinal fluid (graph 2) as found at lumbar puncture does not essentially

*In one case simultaneous determination on the subdural fluid and blood serum on the day of operation gave the following comparative figures:—serum protein 6200, serum N. P. N. 28, serum chloride 601; subdural protein 5300, subdural N. P. N. 33, subdural chloride 605. Such figures can only be accounted for by the breakdown of protein within the subdural fluid.

alter the shape of the graph though the findings are less convincing because the cases are fewer in number. This is likewise true (graph 3) when the cases in whom a clot was proved to be present are segregated from all others and that the graph may be false and with added experience may well conform to the shape of the others. Such as it is however, this graph has an initial rise that is so abrupt as to be almost perpendicular. In addition the level of


Graph II
Graph of Cases with and without Blood, Cerebrospinal Fluid.
Blood C.S.F.
Clear C.S.F.
Protein in Milligrams per 100 cc.
Days between Puncture and Removal
Graph III
Graph of cases with and without Clot
Clot Present
Clot Absent
Protein in Milligrams per 100 cc.
Days between Puncture and Removal

plotted. Even the high point of the peak is approximately at the same level in all curves.

When the cases in whom at operation a subdural clot was grossly absent are considered, however, there is an interesting variation. To be sure this is only a tentative suggestion because the number of cases involved is so few the peak is some 4000 mg. per cent lower than the others while the low level of slow dilution is reached much earlier. The protein concentration is much less at the start (200 as compared to 1200 mg. per cent), the peak is reached between the fifth and tenth days instead of the fifteenth to twenty-first and the abrupt part of

the dilution curve reaches its point of leveling off at the twenty-first instead of the forty-seventh day. These cases are similar to those heretofore known as "hydrops of the subdural space". My feeling pending further data is that they rather represent a condition in which the subdural space has been filled with a very small amount of hemorrhage and a relatively large amount of cerebrospinal fluid which for want of better explanation we may presume has come there through a tear in the arachnoid membrane. A minimum amount of this blood has clotted but the rest has remained at first mixed with, and then in solution, in the cerebrospinal fluid. Such reasoning is supported by the abrupt rise in the first part of the graph and the lower level of the protein value at the peak. Dilution then proceeds more rapidly because there is no further solution taking place as illustrated by the steeper slope and earlier low point of the second part of the curve. Such cases should have as a class a minimum latent period and if any of the symptoms of increasing intracranial pressure develop, they should be relatively mild in character.

From a consideration of the above clinical and chemical evidence, particularly when viewed in the light of Gardner's experimental work, I believe that we may reasonably look upon subdural hematomata as potentially expanding lesions. The length of the latent period will be related to the amount of cerebrospinal fluid mixed with the blood at the time of the injury, the rapidity of the liquefaction and molecular breakdown of the solid clot, the thickness and nutrition of the organized capsule of the hematoma which surrounds the central liquefied area and the approximation of the latter to a capillary bed. These relationships will be in an inverse ratio, i.e., the greater the amount of cerebrospinal fluid admixture the shorter the latent period, etc. Furthermore under such conditions it is probable that a certain number of hematomata in which the process of repair rapidly outdistances the process of destruction, will remain almost completely solid for long periods, even months or years. On this basis it is evident that the pathological picture of chronic subdural hematoma is but a late variant of the acute phase and that at the late stage the hematoma may be liquid or solid or both depending upon the osmotic relationships that have been formed.

DIAGNOSIS. In my experience the diagnosis of subdural hemorrhage can be made only by exploration. I believe that this procedure is always indicated in those cases in whom adequate treatment for one of the commoner craniocerebral injuries fails to bring about improvement. (Chart III.) To be sure it is inevitable that in

CHART III

DIAGNOSIS

- **Skull**
  - Linear and/or Comminuted Fracture
  - Depressed Fracture
  - Compound Fracture
  - Special Fractures
    - Involving Para-Nasal Sinuses
    - Middle and/or External Ear
    - Including Pneumo-encephalocele
    - Cribriform Plate
    - Bullet Wounds
- **Brain**
  - Concussion
  - Edema
  - Contusion and/or Laceration
  - Special
    - Hemorrhage — Sub or Extra — Dural
    - Meningitis
    - Rarer Forms
      - Sub-Pial
      - Sub-Cortical
      - Intra-Ventricular
      - Rupture of Venous Sinus
      - Cortical Abscess
      - Multi-Petechial
- **Scalp**
  - Lacerated and/or Incised Wound
  - Contusion
  - Hematoma
- **General Bodily Condition**
  - Surgical Shock
  - Exhaustion
  - Dehydration

any such exploratory procedure there will be a certain number of unnecessary operations. In this type of case, however, I believe that a properly conceived procedure carried out in an adequately equipped clinic after the patient is out of surgical shock alters little if at all his chances of recovery should it prove to have been unnecessary. On the contrary if a hitherto unrecognized clot is found, his chances are immeasurably improved. The procedure should consist of a trephination of both squamous temporal bones under local anesthesia with sufficient enlargement of the bony opening to permit of subdural exploration with a brain spoon in all directions. During 1933 such a procedure has been carried out in my clinic fifty-one times. In twelve of these no sub- or extra-dural blood was found. Of these twelve, eight were suffering from a lacerated brain or intracortical hemorrhage of such nature that operative interference could accomplish nothing, but no case so far as could be told succumbed because of the operation.

Subjective Symptomatology. Historical data as compiled from the records of these patients with subdural blood are interesting and suggestive but of little selective diagnostic value. (Chart IV.) A history of injury was practically constant and where it occurred was severe in three-fourths of the cases. Loss of consciousness was even more common, occurring in association with each injury and in addition three times where no such history was obtainable. Headache was definitely absent only once, being definitely present in about half the cases. Its presence or absence was unascertainable in the rest of the cases because of unconsciousness. Such symptoms are common to almost all forms of brain injury however.

CHART IV

SYMPTOMATOLOGY—SUBJECTIVE

	History of Injury	Loss of Consciousness	Headache	Convulsions	Mental Change	Latent Interval
Positive	52	55	32	13	57	15
Absent	8	5	1	45	4	39
No note	2	2	29	4	1	8

Mental changes other than unconsciousness and sufficiently obvious to be recognizable by family or friends were unexpectedly frequent. This appears to be one symptom whose presence definitely suggests subdural hemorrhage. A "latent interval", usually defined as the time that intervenes between the receipt of the injury and the onset of symptoms, has been commonly accepted as an essential finding in this condition. I believe the importance of its presence has been much overrated. This is undoubtedly due to the fact that the conception of the acute subdural hematoma as a clinical entity has been completely overshadowed by the intense study of its late form. Such a latent period occurred only fifteen times in this series. The shortest of these was two hours which occurred twice and the longest two and a half years. The other times were one day three times, two days twice and three, five, six, seven, forty-two, fifty-two and eighty-four days. To be sure there were other cases in whom it was impossible to determine either the time of injury or the time of onset of symptoms, but even with these included the number is only twenty-three or approximately one third of the total. Convulsions have been conspicuous by their absence. Having in mind the location of the lesion this finding has been rather surprising.

We may conclude then, that patients suffering from a subdural hematoma will present a history of injury to the head followed in practically every case by some degree of unconsciousness and very often by headache. Their friends and family will subsequently note peculiar actions and speech which on analysis will be found to be due to abnormal mental activity. If the clot is not too large and the original injury is trivial, a latent period with mild or no symptoms may follow. This may be absent or may vary in length up to years but it need not therefore be concluded that more than the original hemorrhage has occurred. These patients will only rarely have convulsions.

Objective Symptomatology. (Chart V.) This is no more selective than the subjective. However abnormalities are sufficiently common so that if physical and neurological examinations are carried out enough will be found to justify hospitalization immediately after the injury.

The blood pressure was elevated 21 times. This was in each case due to the increased intracranial pressure and subsided again to normal levels after removal of the clot. However it was normal even more often, so that elevation is by no means essential in making the diagnosis. Some abnormality on physical examination pointing to an injury was found in 49 of these patients. Easily elicited signs of alterations within the nervous system were found fifty-eight times. Recognizable mental changes were most common (fifty-three) although signs of interference with the neuromuscular apparatus occurred almost as often (fifty). Paralysis in the cranial nerve fields was present thirty-six times. Twenty-six of these were in the facial distribution. This is evidence of the usual site of the clot. Abnormalities were recognized in the eyegrounds only ten times. An ipsilateral di-

lated pupil has been described by several authors[9, 10] as a reliable localizing sign in these cases. This is contrary to my experience. Twenty-five showed no dilatation at all, fourteen were dilated on the side opposite the hemorrhage, three were dilated bilaterally and fourteen on the ipsilateral side.

We may conclude then from the examination of the patient that if his blood pressure is normal or elevated, his physical and neurological examinations abnormal, if he shows mental changes and has a paralysis of one facial nerve, headache, mental changes, an abnormal physical and neurological examination, a paralysis of one facial nerve and a bloody cerebrospinal fluid under increased pressure and who can subsequently have a latent period free of symptoms, may have a subdural hemorrhage. If improvement of these signs and symptoms does not follow proper treatment by dehydration and lumbar puncture, this diagnosis should be confirmed or ruled out by a bilateral exploratory subtemporal trephine. It is equally true that if, in a patient known to have a lacerated brain,

CHART V

SYMPTOMATOLOGY—OBJECTIVE

Blood Pressure	Elevated	21	Dilated Pupil	Ipsilateral	14
	Normal	33		Contralateral	14
	No notes	8		Bilateral	3
				Not dilated	25
				No note	8
General Physical Examination	Normal	12	Neurological Examination	Negative	1
	Abnormal	49		Motor	50
	No note	1		Mental	53
				Cranial Nerves 36	VII — 26
					II — 10
					VIII — 2
					V — 1
					III — 2
					VI — 1
					XII — 1

he is liable to be suffering from a subdural hematoma. If he has one dilated pupil it is as likely to be on the side opposite the clot as it is to be ipsilateral.

CHART VI

CEREBROSPINAL FLUID

	Pressure at First Lumbar Puncture		Protein Content	
	Bl. or Yellow	Clear	Bl. or Yellow	Clear
Increased	18	16	14	3
Normal	10	2	6	5
Decreased	1	1	—	—
Not done			33	1
Not given	14			

Cerebrospinal Fluid. (Chart VI.) Examination of the cerebrospinal fluid will serve only to identify the presence of an increased intracranial pressure and blood from an associated contused or lacerated brain. A little more than half of these cases had a high intracranial pressure and a few less than half had evidence of subarachnoid bleeding. The protein content was increased only in the presence of fresh or old blood and never approached the levels found in the fluid collected from the subdural space.

On the basis of all the above findings I feel that a patient who has been knocked unconscious following a blow on the head, who has a the neurological signs fail to clear up, unconsciousness deepens, delirium or mania develops, the pulse drops to below sixty and the cerebrospinal fluid gets less bloody even though the pressure may be either high or low, some additional and hitherto unrecognized condition must be present. If study of the fluid intake and output relationships and determination of the sugar and cellular content of the cerebrospinal fluid rules out toxic dehydration and meningitis, bilateral exploratory subtemporal trephine is the only remaining method of disproving the added presence of such a sub- or extra-dural hemorrhage.

TREATMENT. It is unanimously agreed that once the diagnosis is made and the patient is no longer in a state of surgical shock, the treatment of subdural hematoma is exclusively surgical. Opinions regarding the best technical method to use are still divided, however. Coleman[11] who reports the next largest personal series advocates large decompressions in the acute, and osteoplastic flaps in the chronic types. Putnam[12] prefers an osteoplastic flap though recently he has discarded this for a decompression in acute cases. McKenzie[13] has used the flap method in the past but has abandoned it in favor of small trephine holes with tube drainage. Fleming and Jones[14] in eight cases used through and through irrigation between trephine holes placed front and back. There is also a great

variation in opinions relative to the value of complete as opposed to incomplete removal of the clot and membranes. On the other hand drainage, especially in association with decompression, is popular.

I believe that the most effective procedure is the one that is easiest and quickest and that still gives an adequate exposure. In addition if it can be made to, it should be so designed as to be of use during the postoperative period. These criteria are best fulfilled by the ordinary subtemporal decompression. This is true, however, only when the bony and dural opening is extended to the limits of the squamous portion of the temporal bone. This operation can be done easily and rapidly and is centered over the most common site of these lesions. The entire cerebral subdural space can be adequately explored and therefore the entire extent of the clot can be removed, preferably by suction. In addition the degree of tenseness of the scalp over the decompression opening will be of help in the early recognition of an increased intracranial pressure.

Drainage of the subdural space should be practised. This will be necessary, not on account of any secondary hemorrhage, but because it is impossible to completely remove all the abnormal content of the space at the time of the operation. It will not always prove efficient, however, because the tract may be completely closed within a few hours by the swollen cortex. A possible exception to this may be noted if a tube drain is used as recommended by McKenzie.

I believe that the entire clot should always be removed. One of the cases in this series died because this precaution was neglected. If exploration of one side fails to reveal any pathology the other side should be invariably opened regardless of any of the symptoms that have been present. In the presence of a complicating contusion or laceration of the cortex, irrigation with salt solution should be avoided. In two cases a local edema was produced in a previously normally pulsating brain in this way. It developed so rapidly that it interfered greatly with the closure and it was necessary to give hypertonic glucose solution intravenously while the wound was still open to prevent further splitting of the cortex. A very common but not constant complication is a severe edema of the cortex. This may be limited locally and not cause any general increase in intracranial pressure. In such cases lumbar puncture drainage is useless and the large decompression is a great help which I am sure enhances the effect of the dehydrating agents that must be used. If the edema is generalized and the intracranial pressure rises, the safety valve action that is provided is also distinctly useful in association with lumbar puncture drainage. A possible cause of this edema is the overexpansion of the previously compressed brain in an effort to fill the abnormal space left after removal of the clot. In this connection Henschen[4] advocates the intravenous administration of sterile water on completion of the operative procedure. This is an interesting suggestion and quite at variance with all others that have been made for solving this particular problem. I know of no one else who has had experience with it.

In my hands this postoperative edema has been best treated by 50 per cent glucose intravenously, 100 cc. at a time. This dose may be repeated every two hours for five doses if necessary. Caffeine given in the same way is also useful if the respirations begin to fail. It is of no help when given by other methods. As a last resort 200 cc. of a sterile 2 per cent solution of $MgSO_4$ can be given intravenously. This is heroic treatment and dangerous. It must be given slowly and, if efficient, will produce a toxic dehydration which of itself may be fatal. This is only of use if there is an adequate circulation. If hyperthermia develops, cracked ice enemata and warm sponges followed by the exposure of the entire body surface to a gentle steady breeze from an electric fan has accomplished a great deal.

This edema is a most alarming and serious complication. Its successful treatment requires constant attention on the part of the physician and the use of all possible and reasonable procedures. Re-operation is contraindicated however. It should be further noted that as the result of the therapeusis, a toxic dehydration may set in, which in its turn will require active interference. On this account an accurate daily chart of the fluid intake and output must be kept. Of the thirty-nine cases subjected to such operative and postoperative care as outlined above, sixteen died, a mortality of 41.2 per cent. This is of course high, but is not to be considered in comparison with the 100 per cent mortality that follows failure to recognize the frequency of this condition until it is discovered by the pathologist.

CHART VII

MORTALITY

	Total Cases		Mortality Percentage
	Living	Dead	
Without operation	0	22	100%
With operation—Neurosurgical	23	16	41.2%

ILLUSTRATIVE CASE REPORTS

ETIOLOGIC AND PATHOLOGIC VARIATIONS

Rupture of Cortical Bridging Vein. ***No. 23. A case of right temporoparietal subdural hematoma caused by rupture of "bridging cortical vein" following a***

homicidal blow: kindness of Dr. Timothy Leary. This young adult white male was admitted to the hospital on a medical service Sept. 28, 1932 after having been discovered unconscious by the police ten minutes previously. The admission diagnosis was "alcoholism". Subsequent investigation showed that he had been last seen the previous night and was normal in every way at that time. On admission he was in moderate "shock" with flushed face and an odor of alcohol on his breath. Temperature 98°, pulse 90 and respirations 32. Physical and neurological examinations were negative except for abrasions and contusions of the forehead, the lips and mucous membrane of the mouth and the left upper and lower eyelids. Death ensued in a few hours. Autopsy showed a large right temporoparietal subdural hematoma caused by the rupture of a vein which ran from the surface of the temporal lobe to the adjoining dura. There was no fracture of the skull and no injury to the brain.

Lacerated Brain with Cerebral and Cerebellar Hematomata and Bloody Cerebrospinal Fluid. *No. 58. A case of right cerebral subdural hematoma together with a cerebellar subdural hematoma caused by the tearing of cortical vessels in association with contusion and laceration of the brain; operative removal of the cerebral hematoma. Death eleven days later with discovery of the cerebellar hematoma at autopsy.* This thirty-eight year old white male who was said to be a "bleeder" was admitted on October 27, 1933 with the history of having gone on a "drinking party" nine days before. The following day he was stuporous and restless. Lumbar puncture showed bloody cerebrospinal fluid under a pressure of 280 mm. water. With repeated punctures for the succeeding six days, he at first improved and then gradually lapsed into coma again. On admission his temperature was 101.4°, pulse 84, respirations 25 and blood pressure 132/102. There was a dilated right pupil, a spastic left arm and a weakness of the entire right side. There was a bilateral ankle clonus and dorsiflexion of both great toes. A right subtemporal decompression was made and through it a liquefied subdural clot, completely surrounded by membrane and covering practically the entire right cerebrum, was removed. The fluid portion showed 7724 mg. per cent of protein and the membrane was that of a "subdural hematoma with well organized new membrane formation". The patient improved for twenty-four hours and then lapsed back into coma. The intracranial pressure rose to and remained around 250 in spite of seven lumbar punctures. As a result on Nov. 1, 1933 an exploratory left temporal trephine was made. The brain was flattened and did not pulsate but was otherwise normal. Puncture of the lateral ventricle showed clear fluid under abnormally high pressure, the removal of which was followed by pulsation of the brain. Unconsciousness continued, however, to be followed by bouts of hyperthermia and a left-sided convulsion four days later. Two days later hypostatic pneumonia and death ensued. Autopsy showed a bilateral cerebellar subdural hematoma.

Cerebral Subdural Hematoma with Clear Cerebrospinal Fluid, an Ipsilateral Hemiparesis and Epilepsy. *No. 46. A case of left fronto-parieto-temporal subdural hematoma caused by bleeding from an unidentified source following an injury sustained during an epileptic seizure. Bilateral subtemporal exploration with left decompression and removal of a fronto-parieto-temporal subdural hematoma.* This twenty-seven year old white single male, who was known to have had generalized epileptic convulsions for at least ten years was admitted for the third time in three years on September 12, 1933. The epilepsy has remained stationary for three years except for a moderate slowly progressive mental deterioration. Twenty-six days before admission he fell striking his head. A rather constant occipital headache, nausea and vomiting and increasing drowsiness followed. Examination revealed a vertical nystagmus, a left hemiparesis including the face, a tendency to hold the right arm in a catatonic posture and a dry skin and tongue. There were no abnormal reflexes but there was a slight irregularity of the left pupil. Lumbar puncture showed clear fluid with no cells under a pressure of 140 mm. water. On September 12, a right-sided subtemporal exploratory trephine revealed a normal brain under increased pressure with marked edema and flattening. A left-sided subtemporal decompression disclosed a clot over the anterior two thirds of the hemisphere lying in the subdural space and solid except for a liquefied area in the centre. This was removed including the membranes. The patient made an uninterrupted recovery and was discharged on Sept. 25, 1933, 13 days after admission. Microscopic examination of the membrane showed a well-organized subdural hematoma and chemical examination of the fluid part of the clot yielded a protein of 5340 mg. per cent.

A Combination of Cerebral Subdural Fluid on the Right and a Cerebral Subdural Hematoma on the Left with clear colorless Cerebrospinal Fluid. *No. 8. A case of right-sided cerebral subdural fluid without clot in combination with a left-sided cerebral subdural hematoma following an injury sustained playing football. Drainage of the right subdural space with decompression; attempted ventriculogram, which was followed twenty-five days later by removal of a right frontotemporal subdural hematoma and a right-sided decompression. Recovery with permanently increased intracranial pressure.* This eleven year old white boy was referred by Dr. Chute of Osterville and admitted on December 8, 1932. He was injured during a football game on November 17, 1932 but was never unconscious. During the following week he developed a headache though he continued at school. Subsequently his headache grew worse, he vomited, became semiconscious and was disoriented. The physical and neurological examinations were negative except for his mental state, an early choking of the discs, a weakness of the left external rectus muscle and a right pupil that was larger than the left. Lumbar puncture yielded clear fluid under a pressure of 300 mm. water with protein of 27 mg. per cent and a sugar of 87. On January 1, 1933 a right subtemporal decompression was made and a large amount of clear yellow fluid removed from the subdural space. The brain was compressed and the arachnoid firmly plastered against the pia. No clot was visible anywhere. The fluid was neither measured nor examined. Following this procedure he was symptomatically better but continued to run an intracranial pressure of about 250 mm. water, in spite of almost daily lumbar punctures. On January 20, ventriculogram was attempted but the films were unsatisfactory. Lumbar punctures were continued without further improvement until February 14, 1933. On this date a left subtemporal decompression was made and through this a thick-walled cyst covering the temporal lobe was emptied of one and one fourth ounces of thick serum-like fluid. The collapsed cyst wall, which was unquestionably that of an old organized hematoma was then removed. Microscopically the cyst wall was that of a well-organized subdural hematoma. The fluid contained 666 mg. per cent of protein. Following this the patient's symptoms and signs completely cleared up except for a persistent elevation of intracranial pressure to about 250 mm. water. In spite of this he was discharged symptom-free on March 5, 1933.

Cerebral Subdural Fluid with a Lacerated Brain

and Bloody Cerebrospinal Fluid but no Hematoma. *No. 37. A case with a collection of brownish yellow subdural fluid over the left cerebral hemisphere associated with a laceration of the brain following an automobile accident. Bilateral exploratory trephine with left decompression and drainage of the left subdural space. Recovery.* This thirty-five year old white male was admitted to a general surgical service on April 12, 1933 following an automobile accident. He was unconscious and remained so for two days. There was a compound fracture of the left frontal region, bloody cerebrospinal fluid under a pressure of 450 mm. water, a left facial paralysis and a pulse between 50 and 60. He was irrational, delirious, restless and apparently aphasic. On April 18 he was transferred to the neurosurgical service on account of deepening coma and failure to improve under dehydration and repeated lumbar punctures. After a period of observation and treatment to rule out the possibility of toxic dehydration a left exploratory subtemporal trephine was made and enlarged to a decompression. Through this opening a large quantity of thick yellow brown fluid escaped from the subdural space. This fluid contained 4800 mg. per cent of protein. After its removal it was possible to survey the lateral surface of the entire hemisphere. This was stained a deep chrome yellow. The subarachnoid space which at first was completely empty gradually refilled, and a small laceration of the surface of the temporal lobe was discovered; but nowhere could either a hematoma or the membranes of a liquefied hematoma be found. A similarly placed exploratory trephine was made on the right side but the brain and its membranes were normal. His signs and symptoms completely cleared up and he was discharged relieved May 8, 1933, 24 days after admission.

Cerebral Subdural Hematoma with a Protein Content of the Liquefied Clot almost Double that of Normal Blood Serum. *No. 49. A case of right cerebral subdural hematoma from an unknown source which developed following a recent drinking bout in a chronic alcoholic who also had cardiovascular syphilis. A bilateral subtemporal decompression with evacuation of a partially liquefied subdural hematoma was followed by relief of symptoms and discharge eleven days after operation.* This fifty-two year old white male was admitted on a medical service on October 19, 1933 because of vomiting and stupor. He had previously been admitted to the alcoholic ward several times. He stated that he had been drinking continuously for five days and that for three days he had been feeling depressed, and had had a pain in the back of his head. This pain followed a fall down two steps which occurred three weeks previously. He presented a right-sided weakness, bilateral Babinskis, thick mumbling speech, incontinence and irregular but equal pupils. He had a blood pressure of 172/102 with an enlarged heart with a systolic murmur at the apex, aortic area and neck. His blood Kahn was positive. On October 20, 1933 he was transferred to the Neurosurgical Service and on October 21, 1933 bilateral subtemporal decompressions were made, the brain on the left being flattened and edematous. On the right there was a partially liquefied "currant jelly" subdural hematoma covering the entire lateral aspect of the hemisphere. No laceration or other source of the hemorrhage was found. Following this operation and a period during which he was in surgical shock his symptoms and signs cleared up, his intracranial pressure which before operation was 350 mm. water with clear fluid fell to 170 mm. water and he was discharged relieved on November 1, 1933 ten days after operation.

Unusual Subjective Symptomatology

A Fatal Case of Extensive Acute Cerebral Subdural Hematoma with no History of Injury. *No. 44. A case of large left cerebral subdural hematoma following one hour after swimming and the ingestion of a heavy meal, and associated with acute vascular hypertension. Operative removal through a left subtemporal decompression followed by rapidly advancing postoperative cerebral edema, circulatory failure and death.* This fifty-nine year old white male was admitted to a medical service on August 2, 1933 with a history of headaches for three to four years from an undetermined cause and with a known systolic blood pressure for the past year of only 135. Ten days before admission, after swimming and a heavy meal, he had a severe headache and fainted. It was categorically stated that he had had no fall or other injury at any recent time, nor before, during or after swimming. On examination he was stuporous with a blood pressure of 220/110, a dilated left pupil, early choking of the discs, a bilateral Babinski and a left-sided weakness including the tongue and face. Lumbar puncture showed a xanthochromic fluid with eight lymphocytes and no red blood cells, under a pressure of 250 mm. water. He was transferred to the Neurosurgical Service the following day and operated upon at once. A left subtemporal decompression was made and a "currant jelly" subdural clot which covered the entire lateral surface of the hemisphere was removed. This was thickest and most adherent over the occipital cortex but no source for the bleeding could be found. One hour after operation his temperature rose to 102 and his pulse dropped to 60. The blood pressure was only 70/40. A lumbar puncture showed a cerebrospinal fluid pressure of 700 mm. water which fell to 30 following the removal of 10 cc. of yellow cerebrospinal fluid. Following this and the administration of 150 cc. of 50 per cent glucose intravenously his pulse rose to 80 and blood pressure to 155/100. One and one half hours later a similar collapse occurred. Two hundred cc. of 2 per cent magnesium sulphate solution was given intravenously with slight general improvement and a rise of blood pressure to 130/90. However circulatory failure set in, his lungs filled up and he died twelve hours after the operation. Autopsy showed no further subdural bleeding.

Cerebral Subdural Hematoma in a Patient Who Had Been Committed to a Psychopathic Hospital as "Alcoholic Psychosis" and Who Completely Recovered Following Removal of the Hematoma. *No. 62. A case of right cerebral subdural hematoma following a fall on the street. Development of mental symptoms that led to commitment to a psychopathic hospital for an observational period. Admission on medical service as lead encephalopathy. Operative removal of right cerebral subdural hematoma with discharge free of symptoms seventeen days later.* This forty-seven year old white male house painter was transferred from the medical service to the neurosurgical service November 22, 1933. He was a known chronic alcoholic. On October 20, 1933 he was brought home drunk following a fall on the street which had caused a laceration of his scalp. The following day he was sent by the police to a psychopathic hospital on account of disorientation and confusion. During the following two weeks at this hospital he became increasingly stuporous. Stippling was found once in his red blood cells and following the development of a left hemiplegia he was transferred to the Boston City Hospital on November 20, 1933 as a possible lead encephalopathy. On the medical service the stippling could not be confirmed but a lumbar puncture yielded yellow fluid under a pressure of 200 mm. water. He did have an anemia which, however, proved to be due to

fifteen years of bleeding hemorrhoids. On transfer to the Neurosurgical Service he was in coma with a flaccid left hemiplegia, hypertonic right arm and leg, bilateral hyperactive tendon reflexes, a right Babinski and a dilated left pupil. His blood pressure was 154/94. A right subtemporal decompression was made and a partially liquefied subdural hematoma covering the frontotemporal region was removed. The subarachnoid space contained yellow fluid and the cortex was colored dirty brown and was compressed. The fluid clot contained 10691 mg. per cent of protein and microscopic examination showed the solid clot to be that of a well-organized subdural hematoma. He was discharged to the surgical side for treatment of his hemorrhoids free of all other signs and symptoms seventeen days later.

Subdural Fluid, Abnormal Bridging Vein, and Epileptiform Seizures after a Four Year Latent Period. *No. 34. A case of right cerebral subdural fluid associated with delirium after a four year latent period. Exploratory craniotomy with drainage of the subdural space, division of an abnormal "bridging vein" and relief of symptoms to date for one year.* This thirty-one year old white male was admitted on April 1, 1933 because of attacks of headache associated with delirium and unconsciousness. These have been present at infrequent intervals for the past one and a half years. Six years ago he was unconscious for two hours following an automobile accident in which he sustained a lacerated wound of the scalp. On admission he stated that he was unable to work on account of his attacks. Neurological and physical examinations were negative except for a scar in the right parieto-occipital region and hyperesthesia and atopognosis on the left. During an attack in the hospital he complained of numbness of the left side, inability to move his left leg and weakness of his left arm. Lumbar puncture was not done. On April 17, 1933 a right parieto-occipital bone flap was turned down exposing a dura that did not pulsate and from beneath, while from the subdural space a considerable quantity of yellowish fluid was collected. This had a protein content of 140 mg. per cent. After the subdural space was emptied the brain began to pulsate and exploration of the cortex disclosed a large venous connection between the dura and the posterior parietal subcortical region which anchored the cortex at this point. This was divided and the flap replaced. The patient made an uninterrupted recovery except for a slight necrosis of the posterior medial edge of the scalp flap and has remained free of attacks to date, having been discharged from the hospital ten days after operation.

Subdural Hematoma Associated with Increasing Irrationality and Disorientation Culminating in Active Delirium and Complicated by Extreme Therapeutic Toxic Dehydration following Operation. *See under postoperative complications, case No. 10.*

Unusual Objective Symptomatology

Right Hemiparesis and Hemianesthesia With Choking of Right Disc But Without Any Speech Difficulty. Removal of Subdural Hematoma from the Floor of the Left Middle Fossa With Subsequent Discovery that Patient Was Left-Handed. *No. 33. A case of left temporal subdural hematoma following an automobile accident. Left subtemporal decompression and removal of the hematoma after patient had failed to improve under lumbar puncture and dehydration therapy with complete relief of symptoms.* This twenty-six year old white male was injured in an automobile accident on March 31, 1933. He was unconscious and had a lacerated wound of the scalp. Following eight lumbar punctures in six days he regained consciousness but complained bitterly of a severe headache. These punctures all showed bloody fluid under pressure varying from 450 to 250 mm. water. On this day he was admitted to the Neurosurgical Service, being referred by Dr. B. Gallagher of Waltham. Examination showed an apathetic man with slight right-sided weakness and right-sided hypesthesia. There was very early choking of the discs. The blood pressure was 120/40. The following day a left subtemporal decompression was made and through this a considerable quantity of bloody subdural fluid removed. This was not examined chemically. Because this failed to give the expected relief of the intracranial tension a further search was made and a large subdural clot evidently originating from a lacerated area in the adjoining cortex was found lying on the floor of the middle fossa beneath the temporal lobe. This was removed with return of the intracranial pressure to normal. The patient was discharged relieved and free of symptoms eighteen days after operation.

Decreased Cerebrospinal Fluid Pressure in a Case of Bilateral Cerebral Subdural Hematomata. *No. 57. A case of bilateral cerebral subdural hematomata associated with contusion of the brain and unilateral dilation of the pupil following an injury during a period of acute alcoholism.* This fifty-one year old white male was admitted to a general surgical service in what was believed to be an alcoholic stupor on October 13, 1933. He had a lacerated wound of the scalp which was sutured. A lumbar puncture was reported as showing normal fluid under normal pressure. The next day he was conscious, and was up and about the ward but was noticed to be mentally abnormal. He was restless with irrelevant speech and tended to avoid the other patients. Sixteen days after admission he suddenly became deeply unconscious. A lumbar puncture showed a pressure so low as not to be measurable though old bloody fluid was obtained by suction. Examination showed a dilated right pupil, a bilateral spasticity of the extremities with increased reflexes, ankle clonus and Babinskis. The blood pressure was 150/95 and the pulse 88. A second lumbar puncture eight hours later was similar to the previous one. This hypotension was believed to be due to dehydration from his failure to drink enough liquid. Blood and spinal fluid Kahns were negative. He was transferred at once to the Neurosurgical Service and operated within an hour. A left subtemporal exploration was made with the resultant escape of yellow fluid and clots from the subdural space with a resumption of pulsation of the cortex. A similar exploration was carried out on the right and enlarged to the size of a decompression. Through this was removed an encapsulated partially liquefied subdural hematoma which covered the entire lateral surface of the hemisphere. The liquid portion had a protein content of 8,510 mg. per cent and microscopic examination of the walls was reported as showing an organized subdural hematoma. The brain was contused. He remained unconscious until his death. Two days after the operation he developed left-sided convulsions and four days postoperatively a lumbar puncture showed a pressure of 125 mm. water. The convulsions continued, his circulation began to fail and he died from this latter cause on November 4, 1933, six days after operation. Autopsy showed his brain damage to be much more extensive than had appeared at operation with no further significant subdural bleeding. The cause of death was given as brain injury.

See also for Ipsilateral Hemiplegia under "Etiology and Pathology" Case No. 46 and under "Subjective Symptomatology" Case No. 44.

Therapeusis

Right Cerebral Subdural Hematoma Not Found Because of Failure to Explore Both Sides of the Skull.

No. 43. A case of right cerebral subdural hematoma associated with sudden unconsciousness during imprisonment for a charge of drunkenness. Left subtemporal exploration—nothing found. Death. Subdural hematoma on right. This sixty-seven year old white male was transferred from the jail on August 8, having been imprisoned seven days previously on a charge of drunkenness. He had been found unconscious on the floor of his cell after having been reported as well two hours previously. On admission he was deeply comatose with Cheyne-Stokes respiration, spasticity of all extremities with hyperreflexia, Babinskis and ankle clonus. There was an old bruise on his face. Lumbar puncture yielded bloody fluid under a pressure of 120 mm. water. The blood pressure was 220/120, pulse 100 and of poor quality. A left temporal exploratory trephine was made which showed nothing but a tense edematous, non-pulsating brain. The right side was not done as it was hoped that his condition would improve and he could be again operated upon later. His condition continued to get worse however, and he died without regaining consciousness three days after operation. Autopsy showed a large right cerebral subdural hematoma.

Death Due to Primary Incomplete Removal of a Right Subdural Hematoma. *No. 41. A case of right cerebral subdural hemorrhage from a contused brain which was operated upon twice, the removal having been incomplete after the first interference. Death followed the second operation.* This thirty-eight year old white male was brought to the hospital by the police because of presumed intoxication and a scalp wound on July 16, 1933. The wound was sewed up and he was sent home with his brother. He was brought back in deep coma the following day and admitted to the general Surgical Service. Two lumbar punctures showed bloody fluid under a pressure of 175 mm. water. He was delirious and required restraint. He was transferred to the neurosurgical service at once and operated upon within three hours. A small right subtemporal decompression was made and a clot removed from the exposed subdural space. The left side was also explored but no pathology was found. He was markedly improved for two days followed by a rise in intracranial pressure, renewed stupor and respiratory irregularity. He was at once taken to the operating room and the left side reopened with negative findings. The right side was then reopened and the decompression and dural opening enlarged. This permitted more complete exploration and resulted in the discovery and removal of 30 to 50 cc. additional clot that had been missed at the previous exploration. The patient continued to go downhill and died five hours after the operation from acute cerebral edema and circulatory failure. Autopsy revealed no new pathology.

Postoperative Complications

Acute Rapidly Advancing Postoperative Edema—*see section on "Subjective Symptomatology" case No. 44.*

Therapeutic Postoperative Dehydration Made Necessary by the Occurrence of Acute Postoperative Edema. *No. 10. A case of right cerebral subdural hematoma resulting from a contusion of the brain caused by a fall. Operative removal, rapidly advancing postoperative edema, therapeutic toxic dehydration, recovery.* This thirty-five year old white man was admitted to the neurosurgical service on Feb. 21, 1933. He was injured on Feb. 10, 1933, following a fall of fifteen feet onto a concrete floor. He was unconscious for nine hours during which time he was taken to another hospital. On regaining consciousness his head was x-rayed, found to have no visible fracture, following which he was discharged to his home on Feb. 12, 1933 with a diagnosis of "concussion". Since then to the present he had had increasingly severe occipital headache and increasingly frequent and prolonged periods of irrationality. On admission he showed early choking of the discs, nystagmus, stiffness of the neck and increased tendon reflexes on the left and dilated left pupil. Lumbar puncture revealed bloody fluid under a pressure of 260 mm. water. For three days he became progressively more disoriented and delirious and finally reached a stage where it was almost impossible to control his activities. On February 25, 1933 a bilateral exploratory subtemporal trephine was made, the left side being done first. The brain was not pulsating but otherwise no pathology was found on this side. On the right side the trephine hole was enlarged to a decompression and a small temporoparietal "currant jelly" subdural clot together with a considerable quantity of yellow subdural fluid was removed. The source of the bleeding could not be found. For twenty-four hours following the operation he was much improved but the next day he began to get restless and delirious again. At this time his temperature got to 102° and it was noted that his fluid intake had ranged between 1750 and 3000 cc. in twenty-four hours. His intracranial pressure was normal and his decompression flat and pulsating. On March 3 his fluid intake was raised to 7500 cc., the two preceding days having been pushed to 5000 and 6500 respectively. Coincidentally he became coöperative, conscious with a temperature of 100° and ceased being incontinent. Two days later his temperature was normal, he was oriented and mentally clear, his fluid intake having in the meantime been kept between 6000 and 6500 cc. in twenty-four hours. From that time his progress was uninterrupted and he was discharged relieved and symptom-free March 21, 1933, twenty-five days after having been operated upon.

Summary and Conclusions

1. Subdural hematoma occurs in about one out of every six cases of craniocerebral injury.

2. Subdural hematoma may be an expanding lesion. In such cases there may be a latent period during which symptoms are absent until the intracranial pressure begins to rise above normal.

3. The signs and symptoms caused by subdural hematoma are the same as those caused by any other major brain injury. Patients suffering from the latter get better under appropriate non-operative treatment. Those suffering from the former condition, however, in the large majority of cases die unless operated upon.

4. The certain presence of a subdural hematoma can only be determined by bilateral exploratory subtemporal trephine.

5. Treatment of subdural hematoma can be best carried out through a subtemporal decompression. The entire clot should be removed, drainage should be provided and irrigation in the presence of a contused or lacerated cortex should be avoided.

6. Postoperative edema is a common and serious complication. Treatment should be early and vigorous and include dehydration, lumbar puncture drainage, caffeine and two per cent $MgSO_4$ intravenously, and appropriate measures to lower the body temperature.

7. Without surgical interference the mortality of twenty-two proved cases of subdural hematomata was 100 per cent. With operative treatment as outlined the mortality in a series of thirty-nine cases was 41.2 per cent.

8. The artificial segregation of certain of the so-called chronic forms of this disease should be abandoned. It has come about only because of our failure to recognize the same condition at the time of its original production.

9. A bibliography covering the ten-year period from 1924 to 1934, and a group of typical case histories are appended.

I am happy to have this opportunity to gratefully acknowledge the stimulus and assistance I have received from Dr. Timothy Leary, Medical Examiner of Suffolk County, Boston, Massachusetts. Without his stimulus the work would never have been undertaken; without his help it could never have been completed.

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DISCUSSION

DR. SAMUEL C. HARVEY, New Haven, Conn.: I am glad to have the opportunity of discussing this most important paper of Dr. Munro's.

We have been accustomed in the past, I think, to segregate subdural hematomata into two rather sharply defined groups; the one showing the acute middle meningeal hemorrhage syndrome, the other the so-called chronic subdural hematoma with which the patient returns to the surgeon with a cystic type of lesion.

The point of Dr. Munro's paper which I take to be peculiarly significant is the fact that there is an intermediate or bridging type of hematoma which should be picked up in the acute phase and treated at that time. This is very easily said, but done with much more difficulty.

Judging from his charts as well as from my own experience, the signs and symptoms of this condition are very variable and transitory. They cannot be built up into a clear-cut clinical picture. One must depend to a great extent on the course of events. If the patient is improving and continues to do so, he is probably not suffering from a serious subdural hemorrhage. On the other hand, if he does not improve promptly or if his progress is downhill, then he very likely is. Even then, it is difficult to differentiate this condition and the severe "commotion" (if I may use the term) of the brain which may likewise carry the patient progressively downhill.

It is true without doubt that all of us have overlooked the more acute phases of the subdural

hematoma, and it is likewise true that instances of local injury coming to necropsy show a disturbingly high ratio of subdural hemorrhage. Whether all these patients die from such hemorrhage is another question. In many, the underlying fatal pathology is the severe primary injury to the brain itself.

Nevertheless, Dr. Munro's paper is a very enlightening contribution and should assist us in discovering a considerable number of these lesions and subjecting them at once to operative treatment.

DR. GEORGE A. MOORE, Brockton, Mass.: Gentlemen, I am not going to attempt to discuss Dr. Munro's very interesting paper. This is a specialized zone of surgery that I have felt is not at the present time in the field of the general surgeon. It has been my pleasure to hear Dr. Munro discuss this subject once before at the City Hospital and at the same time see Dr. Timothy Leary's series of brains with hematomata that were found at autopsy.

I am reminded of what Dr. Harvey Cushing said at one time when he was giving a clinic on acromegaly. He stated that it was only a comparatively few years ago that the field of thyroid surgery was limited to a very few specialists in that line of work, but we now see thyroids operated upon by a great many men doing general surgery. He predicted that in a few years we may see surgery of the pituitary gland taken over by the general surgeon.

At present many men in general surgery are not operating on subdural hematomata. Dr. Munro has placed special emphasis upon the number of unrecognized cases with this condition which have been observed at the City Hospital. It would appear that the general surgeon should be more on the alert in the care of cases of head injury to recognize symptoms of intracranial hemorrhage.

Of especial significance in Dr. Munro's statistics was the large number of patients with abnormal physical signs. There were some thirty-eight patients with motor symptoms and about forty patients with mental symptoms. With more painstaking study we should recognize these symptoms in more cases and get these patients into the hands of the neurosurgeons in time to decrease the mortality that now exists.

DR. W. J. MIXTER, Boston, Mass.: I think this work of Dr. Munro's is very valuable. He has created a lot of interest in the subject which has been reflected, not only among the people who are perhaps more interested in neurosurgery, but also, I think, among the men who are doing general surgery.

I disagree with Dr. Moore a bit. I think the general surgeon has to handle a number of these cases because many times the general surgeon will find a case of subdural hematoma among the acute cases in his practice.

We have planned at the Massachusetts General to have our general surgical house officers see the traumatic neurosurgery so far as we can and not confine it to the neurosurgical house officers, because we feel that the general surgeon must handle a great deal of the traumatic neurosurgery, perhaps not the cases that run along and can be handled as late cases, but the acute immediate surgical emergencies.

So far as type of operation goes, Dr. Munro has well stated that there is a great difference of opinion as to the methods of attack, and I should agree absolutely with him that the subtemporal decompression was the best general method to use. In some instances the trephine opening with evacuation of the fluid blood will work well. I think the osteoplastic flap as a general rule is unnecessary. The subtemporal decompression can be extended either forward or backward and works very well in these cases. It has seemed to me that there were a certain number of the subdural hematomata that were non-traumatic and arose probably from the small aneurysms of the circle of Willis, much in the same way that the spontaneous subarachnoid hemorrhages do. That is probably not nearly so common as the traumatic origin but I believe does occur in certain instances, and I should like to ask Dr. Munro for his opinion.

DR. LUCIUS C. KINGMAN, Providence, R. I.: I have seen a number of war wounds of the head which resembled the cases under discussion. The patients entered the base hospital about three days after injury. They would walk in with apparently a simple scalp wound. However, there would be a history of being knocked out at time of injury usually followed by nausea. By the next day they would complain of a restless night, some headache and nausea. With such a story we would open the skull and under the site of the bullet wound we would find a subdural hematoma and often some disintegrated brain. This would be washed out and all made immediate recovery. The after history is unknown. The indication for the operation was made entirely on the history.

DR. CHARLES L. SCUDDER, Boston, Mass.: I have two thoughts in connection with this valuable and timely paper of Dr. Munro. It used to be considered that the taking of various specialties out of the general surgical field diminished the usefulness of the general surgeon. It seems to me that Dr. Munro has illustrated in this series of cases, as has already been pointed out, that the neurosurgeon specialist is contributing to general surgery data which may be of assistance to the general surgeon; he is not taking cases away; he is educating the general surgeon so that he may adequately and early treat the cases of subdural hemorrhage.

The other thought I have in connection with the paper, and I think Dr. Munro has made a very important communication, is the fact that Dr. Munro stresses the point that for an accurate diagnosis, an exploratory trephine opening often may be necessary. That is a very radical statement to make. It is accurate and true and I believe that it illustrates a distinct advance in the surgery of the head, that an exploration of the skull, of the cranial cavity, should be made under certain conditions for the purpose of making an accurate diagnosis of subdural hematomata. Dr. Munro has contributed in this paper to the constructive advancement of surgery, bringing to the general surgeon accurate information of an hitherto unrecognized rather common lesion. He has thereby made surgery more efficient. The advance of exploratory trephining under the conditions stated is an important step in the surgery of the head.

DR. LYMAN ALLEN, Burlington, Vt.: I should like to speak just a moment about a similar case, and first I want to say that I know nothing about subdural hemorrhages except those due to trauma. I think every general surgeon should realize the importance of recognizing the symptoms of traumatic head injuries, such as slow pulse, high pressure, and so forth.

The case I wish to speak of is that of a laboring man who was lifting a large rock onto a truck. He slipped and the rock struck him on the nose and forehead, knocking him down and stunning him. It broke his nose and bruised his forehead. When he was brought to the hospital, his pulse and his pressure were normal, and continued so for the next few days. The broken nose was fixed and he was walking around, feeling perfectly well, and had been transferred upstairs, which we do with some

of our convalescents. He was about to go home when, at nine-thirty one night, he suddenly became unconscious, with stertorous breathing. His roommate called the nurse's attention to his breathing. His pressure was 238/130 and pulse 122. Repeated lumbar puncture showed bloody cerebrospinal fluid under increased pressure. He died three days later. We probably should have trephined, but did not. Autopsy showed a contusion of the right anterior frontal lobe with softening of the clot and rupture through into the lateral ventricle. There was no infection whatever.

I want to ask Dr. Munro what we could have done if we had trephined and had found the contused frontal lobe with softening of the clot.

I do feel that in these days of traveling sixty miles an hour around corners, the recognition by the general practitioner (to say nothing of the surgeon) of symptoms suggestive of brain injury is important. Such symptoms should be looked for carefully and repeatedly.

I also believe that a diagnostic trephine is probably safer for the patient in many cases than masterly inactivity.

DR. MUNRO: Dr. Allen, your case, in my opinion, gets right to the crux of the situation. There is no question but that most of the head injury cases, particularly these difficult diagnostic problems, are going to be left on the doorstep of the general surgeon. The neurosurgeon or the specialist, the neurologist, is going to see only those who live long enough to let him get to them, those who can afford to pay for a consultation, and those whose families will recognize and coöperate with the general surgeon in recognizing the value of getting in a specialist, whatever that value may be.

On the other hand, there are certain responsibilities that necessarily rest on the general surgeon if he is going to take on this work and I think Dr. Allen's question brings up perhaps one of the most important of those responsibilities. This has to do with the equipment in the operating room.

I operate occasionally outside of Boston, not by preference but by necessity and I have yet to go into an operating room in a city outside of Boston that has been properly equipped to handle efficiently these acute surgical emergencies that occur as a result of head injuries, and the usual most obvious lack of equipment is the lack of some efficient method of suction. That seems like a common or garden variety of thing to have. The hospitals I happen to have seen have been equipped with the tonsillar suction apparatus where the suction is made by a pump run by an electric motor, and about the time when you reach the end of the first hour and a half, and when you really want to use the suction most, all the bearings on the pump and motor freeze and you are left without any suction at all just because you have used it incidentally when you didn't really need it.

I don't believe anybody can handle efficiently such a case as you instance, without suction; on the other hand, with suction you can operate on that type and remove by suction all the soft clot and the contused brain and give these patients a very much better chance of recovery than if you merely open the skull and the dura and leave behind this mass of foreign protein to cause further trouble to the patient's already complicated life.

Dr. Mixter points out that there are cases of subdural hematoma that are non-traumatic. There is no question that that is true. Henschen, whom I mentioned, and who is a Swiss, had quite a list of these cases in his paper. He had combed the literature fairly thoroughly and among other things he mentioned arteriosclerosis and the multiple aneurysms that you spoke of.

I am very much interested to hear of your cases, Dr. Kingman. It is too bad they are not in the literature. Perhaps the Surgeon General and you could get them there, because I think any authentic series of cases of proved subdural hematomata that present a constant history is very well worth while.

My impression from the cases I have seen is that the historical side is a very difficult thing to evaluate and I feel the best that I can do is to say that these cases had a severe injury to the head, and perhaps subdural hematoma should be included among the diagnoses.

I am very much struck by the fact that the more one interests one's self in the problem of subdural hemorrhages, the more subdural hemorrhages crop up. I and my then associates were practically kicked into this investigation by Dr. Timothy Leary, the Medical Examiner. He was finding subdural hemorrhages we had missed over and over again and it finally got to the point where we were so embarrassed, we could not attend his conferences. In self-defense we looked into the matter from the clinical side and, as I say, the more we investigated, the more subdural hematomata were found.

THE CLASSIFICATION OF THE CAUSES OF BLINDNESS

International agreement on the classification of the various causes of blindness was sought by delegates from America, Great Britain, and many European countries at the annual meeting in Paris, May 14, of the International Association for Prevention of Blindness.

That statistics on the causes of blindness which can be understood by laymen constitute an important factor in the prevention of blindness was pointed out by Professor F. de Lapersonne, of the University of Paris, president of the Association.

Dr. Park Lewis of Buffalo, N. Y., vice-president of the Association, urged that, in addition to classifying the causes of blindness, an effort should be made to classify the extent or degree of blindness when vision exists but not in sufficient amount to be of practical use.

More than fifty different etiologic classifications of blindness are listed in a report submitted by Lewis H. Carris, of New York City, managing director of the American National Society for the Prevention of Blindness. "The question of a satisfactory classification of the causes of blindness," said Mr. Carris, "has occupied the attention of the American Committee on Statistics of the Blind for the past three years. The Committee represents the points of view of ophthalmology, public health, work for the blind, prevention of blindness and social statistics. It is interested in social and economic as well as medical facts."

Among the major causes of blindness included in the American report are infectious diseases such as diphtheria, measles, ophthalmia neonatorum, scarlet fever, syphilis, trachoma, and tuberculosis; traumatic and chemical injuries sustained in war, industrial accidents, traffic accidents and surgical procedure; and congenital and hereditary causes.—*Excerpts from The Bulletin of the National Society for the Prevention of Blindness, Inc.*

ADENOCARCINOMA OF THE STOMACH*

Case Occurring in a Man Twenty-seven Years of Age

BY ROBERT L. PATTERSON, M.D.,† AND ROBERT E. GROSS, M.D.†

HOLMES and Hampton[1] have reviewed evidence to show that chronic ulcerations in the prepyloric area of the stomach usually represent carcinomatous lesions. It was the opinion of these authors that all ulcers at this site should be subjected to surgical exploration because of the frequency with which neoplasm is encountered. The following example of carcinoma of the stomach tends to support this belief. This case also illustrates the difficulty which may arise in differentiating a benign prepyloric ulcer and a cancer in this region. Gastric carcinoma has been occasionally reported below 30 years of age and the following report again emphasizes that the lesion may be found in the third decade of life.

History: The patient was a 27 year old white man who entered the Medical Service of the Peter Bent Brigham Hospital on June 28, 1933 with a chief complaint of three attacks of epigastric pain and vomiting during the previous six months. The onset of this illness began January 1, 1933 following excessive indulgence in alcohol. This first attack lasted one week and was characterized by recurring gnawing pain in the epigastrium and beneath the right costal margin which was relieved by vomiting. The pain had no relationship to eating. During this week he vomited all solid food that was taken, but no fresh or changed blood was noticed in the vomitus. His symptoms completely subsided on a diet of milk and bismuth powders. Gradually he discontinued the bismuth and returned to his regular diet.

He was then symptom-free for over three months until he again indulged in alcoholic beverages. Following ingestion of alcohol in April, 1933, the epigastric pain and vomiting returned. Pain and vomiting persisted for several days until he voluntarily returned to a milk diet and bismuth powders. This pain during this attack appeared about an hour after eating and was relieved by vomiting and also by taking baking soda.

He was then in fair health until June 23, 1933. At that time the third major attack began which was similar in nature to those previously described. He was referred to the Hospital because of persistence of symptoms. His stools during the previous six months had occasionally been black. During this half-year period he had lost 25 pounds of weight.

The family and past histories were irrelevant.

Physical Examination: The patient was a well-developed but poorly nourished young man in little apparent distress. Temperature 98.6°. Pulse 92. Respirations 20. Blood pressure 140 systolic, 70 diastolic. Except for evidence of weight loss, the examination was negative. There was no abdominal distention, tenderness, mass, or spasm.

Laboratory Data: Hemoglobin 95 per cent (S). Red cell count 4,500,000. White cell count 10,700. Urine negative. Blood Wassermann negative. Stools had positive benzidine and guaiac tests for occult blood. Gastric analysis showed free acid equivalent to 60 cc. of 0.1 normal sodium hydroxide and total acid equivalent to 100 cc. 0.1 normal sodium hydroxide.

Hospital Course on Medical Service: Cholecystograms by the intravenous method showed a well-filled and normally functioning gall bladder. Gastrointestinal x-ray series were taken. Fluoroscopy showed the rugae in the prepyloric area to be thickened, with an irregularity of the wall at this site. The stomach was low and hypotonic. The duodenal cap filled fairly well and appeared normal in contour and

FIG. 1. Roentgenograph of stomach. The two arrows indicate the raised margins at the edges of the lesion on the lesser curvature. Between the arrows is the crater of the neoplasm. Fluoroscopy showed that no peristaltic waves passed across this prepyloric area.

position. There was approximately a 5 per cent residue in the stomach after six hours. These studies suggested a prepyloric ulcer.

During the twelve days that the patient remained in the hospital he rapidly became symptom-free. For the first seven days a first week Sippy régime was prescribed and, for the remaining period, a fourth week Sippy régime was employed. Free acid was well controlled in aspiration material (free acid 0-5, total acid 7-28). He was discharged July 10, 1933.

Subsequent Course: For the following two months there were occasional recurrences of very mild epigastric distress. He was taking a normal diet but used three to five powders per day. He gained 15 pounds in weight. X-ray examination was repeated on September 12, 1933, by Dr. Merrill C. Sosman. This again showed the irregularity in the prepyloric area, extending up from the pyloric sphincter about five centimeters along the lesser curvature. At the incisura there was a definite filling defect with a central crater (fig. 1). No peristalsis passed through

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this zone. The gastric rugae were enlarged. There was no six hour residue. The duodenal cap was normal. The findings suggested neoplasm rather then ulcer. Exploratory operation was recommended.

Second Hospital Admission: The patient entered on the Surgical Service. Physical examination was negative, except for very slight discomfort produced by deep palpation in the mid-epigastrium.

Operation was performed on September 23, 1933, by Dr. Robert Zollinger. The abdomen was explored through a midline upper abdominal incision. A mass about 7.5 cm. in diameter was found along the lesser curvature of the stomach, extending up from the pylorus. The stomach was entirely mobile. There were no palpable glands along the lesser curvature or in the gastrohepatic ligament. No nodules were felt in the liver or elsewhere in the abdomen. The gastric mass was resected with a wide margin, removing over half of the stomach and about two centimeters of the duodenum. The remaining stomach and duodenum were joined by a Horsley modification of the Billroth I type of anastomosis.

The patient made an uneventful recovery. Gastric analysis on October 18, 1933, 25 days after operation, gave the following results:

	Free Acid (cc. 0.1 N NaOH)	Total Acid (cc. 0.1 N NaOH)	Guaiac Test
Fasting	0	20.5	Neg.
20 minutes after test meal	0	12.0	"
40 " " " "	0	7.0	"
80 " " " "	0	2.0	"
120 " " " "	0	10.0	"
30 minutes after 1 mg. of histamine	3.0	25.0	"

The patient was discharged on October 22, 1933 in excellent condition. When last seen in March, 1934 he was symptom-free and in good health.

Pathologic Examinations

Gross Description: The resected portion of the stomach measured 16 cm. along the greater curvature and 8 cm. along the lesser curvature. Two cm. of duodenum were included with the specimen. The tumor presented on the mucosal surface as a large centrally ulcerated mass 45 mm. in length and 35 mm. in width (fig. 2). The distal margin of the neoplasm reached just to the pyloric sphincter. The margins of the tumor were raised 8 to 10 mm. above the general plane of the gastric mucosa. About one half the circumference of this rim was rounded, while the remaining half was irregular, jagged, necrotic, and hemorrhagic. The crater was 35 mm. long and 30 mm. wide. It was 12 mm. in greatest depth. The deepest portion of the crater projected into the musculature of the stomach wall. The mucosa about the neoplasm was edematous and the musculature was hypertrophied proximal to the tumor. On the exterior of the specimen the serosa was smooth and unbroken. There was no neoplastic invasion of the serosa, the included portion of the gastrohepatic ligament, or the regional lymph nodes.

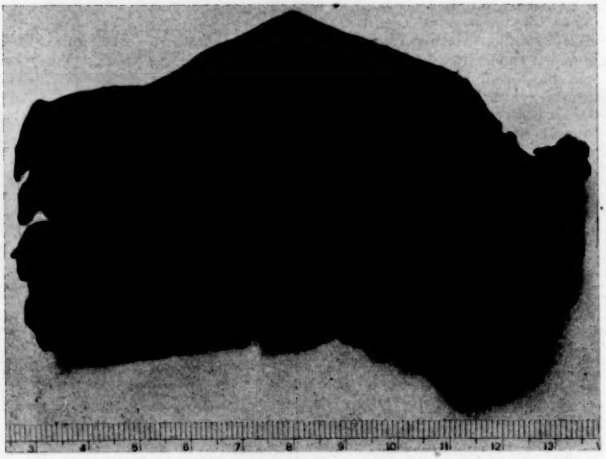

FIG. 2. Photograph of portion of operative specimen. The neoplasm appears with rounded, raised edges and a depressed central crater. The pylorus is at the right of the photograph.

Microscopic Examination: Sections showed an adenocarcinoma. The tumor replaced the mucosa and submucosa and partially invaded the muscularis. There was no involvement of the outer muscular coats, the serosa, or the regional lymph nodes. Where the tumor was poorly differentiated, the cells were large, polyhedral, and varied greatly in size and shape. The nuclei were ovoid, had a fine chromatin material and often contained two or three nucleoli. Most of the tumor was well differentiated and tall columnar cells formed acini and glandular structures of varying size and shape. Mitotic figures were often seen and two or three often could

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be found in a high dry field. The stroma was of only moderate amount. There was a moderate inflammatory reaction throughout the tissue. On the surface facing the gastric lumen there was a fibrinohemorrhagic exudate. The picture was more suggestive of primary gastric carcinoma than a carcinomatous formation at the site of a previous ulceration.

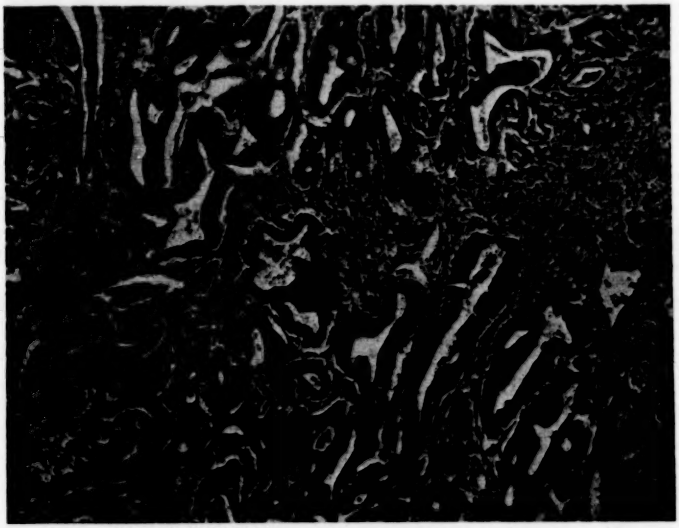

FIG. 3. Photomicrograph of gastric neoplasm × 110. The tumor is here differentiated into acinar and glandular structures.

SUMMARY

Report is made of an adenocarcinoma of the stomach occurring in a man 27 years of age. The symptoms, clinical course, and relief on ulcer treatment suggested that the lesion was of a benign type, but the location in the prepyloric area aroused suspicion of gastric carcinoma. Resection was performed. The patient is in excellent condition six months after operation.

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THE METABOLISM OF LEVULOSE*

IV. The Hepatic Influence On The Utilization of Galactose and Levulose

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IN an earlier communication[21], the authors discussed certain general aspects of the metabolism of levulose as indicated by response to a provocative levulosuria. The present paper deals with the hepatic factor as it influences the utilization of this sugar; for purposes of contrast and amplification, the galactose tolerance has also been measured for the same group of subjects. There is an historic basis for comparing these two sugars as within the last thirty years each has been warmly advocated as the best approach to a measure of liver function and their several claims equally positively denied. Strauss[28] in 1901, influenced by the previous observation of Sachs[22] that dehepatized frogs could utilize glucose and galactose but not levulose, developed a test for liver function

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based upon provocative levulosuria. Four years later, Bauer[2] suggested the substitution of galactose as the test sugar, and during the next ten years a fairly extensive literature, reviewed in part in the paper already cited[21], affirmed and denied the advantages of each sugar as a test for liver function. Yet again, a few years later, the development by Folin[5] and others of more precise methods for blood sugar analysis practically reopened the question, and again a group of reports appeared championing the one as opposed to the other.

Unfortunately, blood sugar curves lack a clarity of outline and are insusceptible of that quantitative interpretation which is the basic criterion of the value of a function test[17-b]. Most recently, a group of communications has appeared canvassing the merits of galactose[10, 3] and the question has been reopened by Shay and his associates[23, 24, 25] reverting to the technique originally outlined by Bauer[2]. Unfortunately, when Bauer first described the test, the carbohydrate paradox (Allen[1]) had not been recognized, and the author set as the evidence of an exceeded tolerance the arbitrary limit of a galactosuria of 3 grams or more with a test meal of 40 grams. Without adding further to the dialectic in this somewhat controversial field, it may be said that within the wide confines of the 3-gram limit, major fluctuations in tolerance can occur and be merged to an uninformative parity of implied normality. Even in the early days of the test, this lack of definition was recognized and the limiting boundary was lowered to 2 grams[15], to 1 gram[31], and in a number of instances[9, 12, 27] to the positive response to a qualitative test. This latter procedure was further refined by lowering the test meal to 30 grams[29, 30] and even to 20 grams[7].

The earlier work, in part at least through a failure to recognize the disturbing influence of the carbohydrate paradox, also ignored the sex difference later described by one of us[17-a]. This has recently been denied by Harding and Moberley[8] on the basis of an essentially different technique. Their results are qualitatively in accord with those of the senior author; they prefer to ascribe the different response of the sexes to a difference in the degree of diuresis which the sugar excites; this would seem to be equally a phenomenon governed by the sex of the subject.

During the past ten years, the senior author and his associates have been studying the metabolism of galactose from a variety of standpoints and a series of reports has appeared in the literature[17]. One of the more recent[17-f] has dealt specifically with the hepatic element as a factor of influence in the metabolism of this sugar. In general, hepatic dysfunction exercises a depressing influence on the utilization capacity of the individual; a certain number, however, without significant complication, showed normal or even increased power to utilize this sugar.

The present study reports the results from a somewhat smaller but equally representative group whose tolerance for both sugars was determined during the course of an elaborate diagnostic study. The technic of administration, the analytical procedures and the needful precautions have already been discussed in the preceding paper[21]. The subjects utilized for the study are recorded in the first table. All of the patients had a demonstrated hepatic dysfunction of some type; in addition, one-half had some complication usually influencing sugar tolerance.

TABLE I

PRIMARY ETIOLOGY

Group			Number
I.	Hepatic Dysfunction, uncomplicated		35
II.	Toxic Pregnancy		22
III.	Thyroid Failure		11
IV.	Miscellaneous		
	Pituitary (dysfunction)	3	
	Ovary (hypofunction)	3	
	Abortion	1	
	Cancer (liver)	2	
	Syphilis	2	
	Psychosis (alcohol)	1	
		—	12
	Total		80

Group I was composed of individuals with demonstrated hepatic dysfunction of varying causes and types. Group II was composed of pregnant women with toxemia, all demonstrating an hepatic factor[19]. Group III was composed of patients with thyroid failure complicated by hepatic disorders, an association of relative frequency[20]. Group IV is a miscellaneous group, half of whom show an endocrinopathy while the other half present other types of morbidity all complicating or associated with a primary hepatic status. These latter were selected as the complications which they present are known to influence the galactose tolerance. The basic data of sex and age are grouped in table II.

TABLE II

SEX AND AGE

Group	I	II	III	IV	Total
Sex:					
Male	10	0	2	5	17
Female	25	22	9	7	63
Age:					
High (yrs.)	71	44	51	60	71
Low (yrs.)	13	20	14	12	12
Average (yrs.)	40	32	37	32	36

Irrespective of the overweighting deriving from the group with toxic pregnancy, there is

a very definite preponderance of females. The age range shows a wide scatter, that of the pregnant women alone exhibiting the narrower boundaries intrinsic in their physiological status. The group averages show a reasonable parity.

Certain other data deriving from history or examination bear more directly on the common factor of the series, i.e., the hepatic element. Nearly 10 per cent of the series had had earlier gall-bladder disease leading to surgical intervention, while double that number at the time of study gave clear-cut evidence of gall-bladder pathology on radiographic examination. In like manner, past or present appendicitis was a common datum and other pathology of the gastro-intestinal tract was reported with significant frequency. Hepatic enlargement was demonstrated in six of the group. Table III presents a convenient summary of these several pertinent features on the basis of relative frequency.

TABLE III

SUMMARY

Datum	%
Gall Bladder	28
Appendix	39
Abdominal and Pelvic Pathology	34
Hepatic Enlargement	8

The several factors susceptible of quantitative expression associated with the carbohydrate exchange fall broadly into three categories, namely, blood, urine, and respiratory exchange. In the first, blood sugar curves, as already noted above, fail to offer a reliable quantitative approach, and with levulose the changes in level produced by even massive test meals are very small. Control blood sugar levels are pertinent, however, as hyperglycemia may produce abnormal urine findings. In the second group, glycosuria as such indicates a disturbance of the sugar metabolism although the factors capable of producing it are protean in their array. The tolerance test, as applied in this study, rests upon evidences in the urine of an exceeded assimilation limit.

The third group of evidences, i.e., the respiratory exchange, has not been undertaken in these studies. Carpenter and his colleagues[4] have shown definite and positive results with both the sugars, and in the case of galactose[4-f] have recorded an interesting difference in the response of the two sexes.

TABLE IV

BLOOD AND URINE RECORD

Datum	Group I	II	III	IV	Total
Glycosuria	29%	23%	9%	25%	24%
Blood Sugar					
High (mg.)	123	102	105	113	123
Low (mg.)	69	70	85	65	65
Average (mg.)	94	86	95	96	92

The influence of thyroid failure in raising sugar tolerance is suggested by the less frequent occurrence of glycosuria in this group (III). The low blood sugar average of Group II reflects the downward trend known to be associated with pregnancy. The hypoglycemic levels of the low records in Groups I, II, and IV may be interpreted in terms of the liver influence; the complementary high figures may be traceable to the emotional reaction to venipuncture, always a source of potential error. As it is, no significant hyperglycemic levels are recorded and the averages fall well within the strict range of the normal.

One special approach to the estimation of the levels of hepatic activity is found in the measurement of bile production following the technique of McClure and his associates[11]. A few of the patients were unable to take the duodenal tube and so the record for this evidence falls a little short (14%) of completeness.

TABLE V

LIVER FUNCTION (MCCLURE)

Datum	Group I	II	III	IV	Total
Number examined	29	20	10	10	69
Color:					
Brown (%)	14	5	40	0	13
Yellow brown (%)	4	10	10	30	10
Green (%)	10	5	0	10	7
Yellow (%)	72	80	50	60	70
% Below normal:					
Furfurol Index	86	80	70	100	84
Cholesterol	59	45	30	60	51
Pigment I	79	85	80	90	83
Pigment II	90	90	70	90	87

Of the four constituents measured, cholesterol is the least influenced by abnormal bile production. Further, low values are less frequently recorded in Group II where high blood cholesterol constitutes a characteristic feature of the basic condition[26], and in Group III where a similar condition may be regarded as possible[12]. So far as the biliary function of the liver is concerned, these figures leave no doubt as to significant hepatic abnormality in the members of this series. Other evidences, omitted in the interest of space conservation, are wholly supporting.

Turning now to the primary matter of this communication, the response of the series to galactose will first be considered. For purposes of orientation, it may be stated that the normal male tolerance level is 30 grams; while the response of the female is broadly influenced by her sexual status and ranges from 20 grams in the prepuberal years through 40 during the adult period of sexual activity to a slight decline (two-thirds drop to 30 grams) after the climacteric. Pregnancy and menstruation lower tolerance, while castration or severe

functional failure of the ovaries restore the tolerance level to 20 grams. Thus departures with the female can be mutually correlated only by expression in terms of percentage deviations; while the male does not show this fluctuation, for the sake of uniformity, relative values have been adopted throughout. The results of the galactose testing are collected in table VI.

TABLE VI

GALACTOSE TOLERANCE

Datum	Group I	Group II*	Group III	Group IV	Total
Above normal (%)	0	5	0	8	3
Average amount	—	+100%	—	+33%	—
Normal (%)	6	80	36	17	31
Below normal (%)	94	15	64	75	67
±0 to —33%	23%	0	36%	8%	17%
—34% to —67%	43%	15%	18%	42%	32%
More than —67%	28%	0	9%	25%	18%

*Two cases not tested.

While it is generally conceded that disturbed hepatic function has a depressing influence on the galactose tolerance, there is a complete lack of agreement as to the types of liver disorder influencing the test. Many of the reports fail to allow for the carbohydrate paradox, and thus individuals showing a marked depression are designated as normal because their galactosuria falls below an arbitrary value which could be produced in the normal individual only by massive supertolerance doses. In a recent paper by Roe and Schwartzman[16] in which they report the administration of galactose on the basis of one gram per kilogram of body weight, the doses range from 44 to 75 Gm. (average for 10 subjects, 63 Gm.), and the amount recovered in the urine from 0.91 to 2.78 Gm., with an average of 1.67 Gm. Thus with relatively large supertolerance doses not one of the group surpasses the inexacting standard originally suggested by Bauer[2].

While the tendency is usually downward[17-f], some uncomplicated liver cases show a normal and a few exhibit a definitely increased tolerance. Making allowance for all possible superimposed factors influencing tolerance and for potential errors, there yet remained six cases (4 per cent) with normal and seven (5 per cent) with increased tolerance. In the present smaller series of Group I, none of the patients studied showed an increased tolerance, 6 per cent were normal, and the remainder exhibited varying degrees of depression. Pregnancy lowers the galactose tolerance and in the later stages the prepuberal level of 20 grams is uniformly encountered in the normal[18]. In this group, all of the members of which had definite evidence of hepatic abnormality, four-fifths of the entire series were normal, one case showed an augmented tolerance, and only the inconsiderable remainder (15 per cent) gave evidence of a depressing hepatic influence. In Group III the augmenting influence of thyroid failure works in opposition to the usual depression of the liver dysfunction. The result is to give normal tolerances in one-third of the group. Finally, in the miscellaneous group (IV) one case with a bilobar pituitary failure, a condition which exerts the most powerful of known augmenting influences, overbalances the hepatic depression and shows the modest increase of 33 per cent above prediction. Further, two cases are normal and the remainder are all depressed. In other words, of the eighty cases represented, over half of whom present superimposed complications which ordinarily affect the galactose tolerance, 3 per cent are above normal, 31 per cent normal, and the remainder, two-thirds of the entire group, exhibit the depression which is the usual response. The distribution is to be regarded as typical; certain implications will be discussed later in the text.

All of the patients in this series were also tested with levulose, using a sugar of the highest purity, a precaution of fundamental importance in tolerance testing with all sugars. Standards for normal levels with this latter sugar at present lack clear-cut definition, as the total levulose series is but a few hundred in number, and this includes both the normal and abnormal. The very high cost of the sugar, the larger amounts used in testing, the time factor for the complete diagnostic survey, and yet other inhibiting influences have prevented that completeness which is the ultimate goal. Enough work has been done, however, to warrant the establishment of approximate values for normal standards.

TABLE VII

NORMAL LEVULOSE TOLERANCE

(Values are approximate)

Condition	Positive Test Meal
Male, adult	100 Gm.
Female, adult	100-125 Gm.
Female, pregnant	50-100 Gm.

While the values are at best approximate, several features are suggestive. First, there does not seem to be evidence of a clear-cut sex difference with this sugar; it is not precluded and a longer series may reverse the present impression. Secondly, while pregnancy produces a lowering in tolerance, it does not manifest itself so early and the decline is slower; the final relative level, however, compares favorably with that shown by galactose. Since the normal standards cannot be straitly delimited, it has seemed best to report the results of levulose testing initially in terms of the actual sugar dose which first produced a demonstrable levulosuria.

Taking the tentative standards of 100-125

Gm. to embrace both the normal male and female, Group I, the only sub-series without extraneous sugar influencing factors, it is evident that 9 per cent are above and 71 per cent below the normal zone. The figures do not compare well with those from the galactose test where 94 per cent were depressed and but 6 per cent normal. Group II, with an even larger zone defining the normal, practically duplicates the distribution in the galactose test. Even if the 100-gram response were interpreted as an increased tolerance, 68 per cent of the series would still fall in the normal zone. In Group III, with the augmenting thyroid influence, 18 per cent show high levels and 18 per cent normal; together they balance the 36 per cent who were normal with galactose. In the final sub-group (IV), there are no cases to parallel the report of increase with galactose; the normal fraction is the same and the depression is more frequent with the levulose than with its fellow.

TABLE VIII

LEVULOSE TOLERANCE

Positive Test Meal	Group I	Group II	Group III	Group IV	Total
Over 150 (%)	3	—	18	—	4
150 (%)	6	—	—	—	3
125 (%)	*6	5	*9	*8	6
100 (%)	14	*9	9	8	11
75 (%)	43	27	46	42	39
50 (%)	20	41	9	25	25
25 (%)	8	18	9	17	12

*Probable normal zone in box.

Broadly speaking, there is a rough general parallelism in the indications with the two sugars; the levulose seems possibly less susceptible to the influences of superimposed endocrine states. But this type of statistical approach does not necessarily indicate the concordance or its lack with the individual components of the series. Waiving any attempt at a nice quantitative reading and expressing each test as normal, increased, or diminished, the data can be subjected to a final analytical approach.

TABLE IX

CORRELATION OF SUGAR TESTS

Datum	Group I	Group II	Group III	Group IV	Total
Number	35	20	11	12	78
Agree (%)	69	65	36	67	63
Differ (%)	31	35	64	33	37

Three of the four groups (I, II & IV) show an almost identical division with two cases in agreement for each failing in concordance. The sole exception, Group III, shows the same partition but in the opposite sense. This would seem to confirm the tentative opinion that the endocrine (thyroid) influence is not the same on the metabolism of the two sugars; qualitatively, at least, disturbed liver function seems to have a depressing effect on both sugars but in a slightly more pronounced degree with the galactose (65 per cent against 58 per cent).

DISCUSSION

A brief review of the findings of others may suitably preface the discussion of these results. Nine authors report a total of 525 normal individuals who have been given 100 Gm. of levulose by the oral route; seventy-one, or 14 per cent, were reported positive to this dosage. Many of the subjects were the so-called "hospital normal," and further, as has already been noted in the text, the criteria for positive response are almost as numerous as the investigators. Strauss[28] the original proponent of the test used a qualitative demonstration of melituria, Frey[7] regarded anything less than 0.1 Gm. as normal, i.e., negative, while Wörner and Reiss[32] adopted the more liberal limit of 0.7 Gm. in the urine to define the normal. Our own results would make it seem probable that on the basis of the Strauss criterion the number receiving a positive report would be definitely augmented.

Nine articles in the literature report a total of 217 pregnant women of whom 180, or 83 per cent, were positive with an oral test meal of 100 grams. Within the limitations noted above for the "normals", these figures would seem to be in general accord with our own results.

Turning to the reports on liver function, seventeen papers report 954 cases, of whom 649, or 68 per cent, were positive to 100 grams of levulose. Superficially, there would seem to be a rough agreement with our own figure of 58 per cent; closer analysis develops a definite lack of concordance. With many of the reports based upon a more exacting standard of positive response than our own qualitative index, there are none the less 10 per cent more cases with a positive test than in our own small series. This figure would undoubtedly be still higher if all reports were reduced to the common denominator of the qualitative approach.

Two investigators report a total of 51 cases with 38 positive (or 75 per cent) when the dose was but 50 grams. Omitting the toxic pregnancy group where a superimposed physiological factor is patently operative, 17 in 58, or but 29 per cent, of our cases give a positive response.

The answer for these discrepancies is not far to seek. In a careful review of much of the lit-

erature supplemented by a small group of personal observations, Freund[6] has weighed the relative authority of the two sugars as indices of disturbed liver function. He finds no parallelism in his own results in the relative indications of the blood and the urine with levulose as the test meal. On the basis of all of the evidences, he concludes that levulose yields the more reliable indications when there is significant parenchymatous involvement, while galactose is the more dependable in cirrhotic conditions. Other states, such as jaundice from mechanical causes, syphilitic involvement of the liver and the like, yield results which are entirely indeterminate. In other words, the cases recorded in the literature, on which the quoted statistics are based, presented severe liver dysfunction, the majority of them with jaundice, and were not equatable with the greater portion of the patients in our own series with whom the liver derangement was functional in character and in larger measure unassociated with jaundice at the time of study. The differences recorded are of degree and not of kind, reflecting the severity of liver impairment. We feel that the approach through a series of graded test meals with the definition of that dose for each individual which will produce a transitory melituria, sharpens the significance of the test as an index of functional hepatic derangement. From the literature one would deduce that the differential diagnostic value of both sugars is most in evidence with the patients presenting obvious signs of severe hepatic involvement with jaundice, and even here the weighting rests on a relative and statistical basis. Failure to give due recognition to other factors certainly influencing carbohydrate metabolism is undoubtedly an element in the contradictory reports which comprise the clinical literature on this subject. The lack of uniformity in the conventions of the test and the criteria of positive response have already received comment.

In the series of studies of which this report is but one, we are endeavoring to ascertain, by a standardized technique and uniform approach, both the identity and character of the factors influencing the metabolism of this sugar and, if possible, to give them quantitative significance. The work must be confined to man—the contradictory findings of Sachs[22] and of Bauer[2] are but one illustration of the many forming the basis of this decision. That the mechanism of utilization of galactose and of levulose is not identical, seems to us a warrantable deduction from this and the other studies recorded in the literature. The use of adjuvants such as brandy[3] or beef extract[14] to sensitize and sharpen the test, has but little to recommend it; the problem is sufficiently complex without the addition of further extraneous factors. Such procedures and those in which more than one sugar is used may enhance the differential meaning of the test but certainly do not clarify the underlying mechanisms of utilization. In the present study the only warrantable inference is that hepatic dysfunction tends to influence the utilization power for both sugars with qualitative agreement in two-thirds of the patients studied and with an equal lack of concordance in the remainder. Further, with both sugars a few subjects show an enhanced tolerance where the agencies usually producing this result are not demonstrably operative. Finally, galactose seems to be a slightly more sensitive index than levulose in this series of liver cases; the difference is, however, inconsiderable.

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PATHOLOGICAL DISLOCATIONS OF THE SHOULDER
BACKWARD AND ROTATION DEFORMITY

BY FREDERIC JAY COTTON, M.D.,* AND GORDON MACKAY MORRISON, M.D.*

IN the main these are results of obstetrical paralysis, complicated often with residual muscle pareses, and with the queer bony overgrowths of which the down-hooked overgrown olecranon is the familiar type.

One has learned to free the hindrance of tight pectorals, and J. W. Sever has very well taught us about clearance of the distorted overhang of the acromion.

In occasional cases, however, we are confronted with a persistent back-slip of the head, and in not a few with an inward rotation of the humerus and of the whole arm, not anatomic but physiologic, conditioned probably on long shortened tissues and loose posterior capsule.

For cases of each of these classes we have to-day a remedy worth noting.

As to the backward luxation: Nicola has

The Nicola operation:
1. Biceps tendon (of long head).
2. Biceps tendon emerging from the artificial tunnel into the bicipital groove.
3. Cut ends of biceps tendon sutured.
4. Acromion.

shown us the way, and I think Roberts' modification of his operation is serviceable. Both, and either, are efficient. Here there is no question of absolute anatomic repair. Nicola's operation is not a "restitutio ad integrum", nor need it be. What we want is an effieient mooring, better than the slinging to the acromion with fascia or tendon. The Nicola operation gives this absolutely. The biceps tendon running in its new course from the glenoid edge through the edge of the head to a fixed point in the bicipital groove gives an absolute and permanent fixation against backward displacement.

In the other cases, which are rare, in which some congenital defect of muscle balance gives *forward* luxation or subluxation of the head, we gain by this operation a real and effective mooring.

In the other class, those cases of obstetrical paralysis in which nearly complete muscle regeneration has been achieved, but in which the elbow comes up first with the hand lagging, there is an operation not new, but not familiar enough, that works.

Disregarding all questions of anatomy, for it is *physiology* that concerns us, we may achieve correction by an osteotomy of the shaft just at or below the pectoral insertion, an osteotomy with rotation outward of the arm, below the cut, through something like 60°.

Temporary fixation with a plate is desirable —otherwise contact of rotated fragments is hard to hold. The results are a bit amazing.

Brief data of four cases follow: R. M., female, aged 27: nurse. Congenital shoulder. Seen September 15, 1931. Left arm showed a clean-cut picture of a brachial palsy of the Erb type. No actual paralyses remained, but she was much handicapped. On raising the arm forward it came up elbow first (internal rotation), and there was a very obvious posterior movement of the head of the humerus back to the back edge of the acromion (posterior subluxation). Motion was limited to a point below the horizontal plane.

On the arm being dropped the displacement reduced itself; there was some inward rotation due to a tight pectoral and the acromion was prominent, seemingly hook shaped.

Skiagraphs showed a heavy hooked prominence downward of the end of the acromion; nothing else save general slight lack of bone growth.

Operation proposed and done: Anterior incision as usual. Acromion chiselled across, turned up. Part of pectoral insertion above was divided. A

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typical Nicola operation was done, the divided biceps tendon pulled back up into the joint, brought down again through a tunnel and carried through the front part of the head to the floor of the bicipital groove. The cut ends of the biceps were then firmly sutured together.

Normal recovery was followed by greatly improved (not normal) range of motion forward, outward, and in outward rotation, and was very gratifying. After about 18 months there was an unfortunate recurrence of overgrowth of bone at the front of the acromion, which will need attention. There is and has been no trace of recurrence of the backward displacement.

E. O., female, aged 21, seen April 29, 1933, in consultation with Drs. Russell Sullivan and John Kerrigan. Chronic dislocation of both shoulders anteriorly. This shows a subcoracoid luxation on both sides. The history is curious and not clear. A healthy active girl, rather overplump, always had something queer about the shape of her shoulders but was never concerned about the lack of motion or use. About a month ago, as a result of an electric shock, the right shoulder was dislocated. It was reduced by Doctor Kerrigan. Shortly after, something happened to the left. I rather question the accuracy of her observation. At all events both shoulders were out and irreducible, showing in the x-ray small bones and some thinning of texture. Both heads were subcoracoid, the right more displaced than the left.

Operation on the right side, at Choate Hospital with Dr. Russell Sullivan, May 5, 1933. Hueter incision. Half of pectoral insertion divided. Incision up the biceps groove and in front of the capsule. The tendon was cut, and the proximal portion pulled into the joint. The soft parts were stripped from the edges of the *small* glenoid, but not easily. The dislocation was reduced and motion assured by stretching. A tunnel from the biceps groove through a point on the front edge of the articular surface of the head, was made through which the biceps tendon end was passed from above downward, and both proximal and distal ends solidly sutured together, and to the edges of the bicipital groove. In suturing the capsule, the subscapularis, which was apparently normal, was advanced half an inch.

Second operation: The *left* shoulder was operated on at the Choate Hospital with Dr. Sullivan, June 20, 1933. The usual anterior incision; the head was forward but not so far out as the other had been. The same difficulty in tearing loose of evidently very old adhesions—from the edges of the small glenoid (particularly behind) in order to make reduction possible, was encountered. Then a typical Burrell-Cotton operation was done.

September 27, 1933. The left shoulder shows nearly normal range of motion. The right still shows limitation of near a half in abduction and external rotation. This was more of an operation than was needed on the right. Both are perfectly useful: neither has shown the least displacement of the head.

C. B., female, aged 30, seen June 13, 1931. Since earliest recollection she has had a partly crippled right arm and has had muscle training. This was an obvious obstetrical paralysis, with good return of muscles but with sharp internal rotation of the arm. For example, she cannot use the hand in eating. As always it is hard in this case to evaluate muscle *imbalance*, muscle spasm, and possible skeletal change. The skiagraph showed marked lack of bony development of both glenoid and humeral head. Nothing else. Disregarding all this, osteotomy was advised and done, June 22, 1931, at the Faulkner Hospital. (Cotton and Morrison.) Anterior incision, osteotomy at the pectoral insertion and a four-screw bone plate was used. During convalescence an accidental trauma gave a lifting out of the bone screws from the upper fragment with a bit of outward bowing and excessive callus. The plate was removed August 22, 1931. Despite mischance, the end-result is excellent with restored outward rotation and forward lifting motion.

M. McC., female. Age when first seen 13 years. (Boston City Hospital.) This was an obstetrical paralysis in a particularly vigorous little girl in whom judicious exercise had brought back practically normal muscle action. There was, unfortunately, about 50 degrees of internal rotation, apparently not all from muscle contracture or contraction. At all events, a cross section of the humerus was made through the insertion of the pectoralis major, and the arm rotated out and held in traction in a Murray-Jones splint. On July 22, 1932, eight years later, opportunity came to reëxamine her. She had fortunately been a swimmer and had limbered and developed the shoulder wonderfully. To-day she shows full free motion, nothing objective save a negligible scar.

MEDICAL PROGRESS

ADVANCES IN PEDIATRICS*

BY R. CANNON ELEY, M.D.†

INFLUENZAL MENINGITIS

THIS type of meningitis is looked upon with a great deal of apprehension, as extremely few cases recover. Rivers[1] in a review of 220 cases in 1922 found a mortality rate of 92 per cent and subsequent reviews with the inclusion of additional cases have shown only fractional changes in this figure. Bloom[2] in a very extensive review of the literature both in this country and abroad was able to collect twenty-four cases with recovery out of 302 cases. Jenks and Radbill[3] recently reported thirty-two cases treated at the Children's Hospital at Philadelphia over a period from 1921 to 1929 inclusive with no recoveries. Gibbens[4] in 1931 reported two cases with one recovery.

Ward, Fothergill and Wright[5, 6, 7, 8, 9] in a series of recent papers have made an intensive study of influenzal meningitis from the point of view of producing a therapeutic antiserum, and the study of the mechanism of the action of such a serum. They confirmed the work of early

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investigators showing the relative homogeneity of influenza bacilli. In addition they have shown that virulent meningeal strains are "smooth" in colony form and produce a specific soluble substance. These latter observations were also made by Pittman[10].

The above authors have also observed that the presence of agglutinins in the antiserum did not necessarily connote bactericidal activity. Precipitins in the antiserum, however, accurately parallel the bactericidins. In other words, in order to destroy the meningeal strains of influenza bacilli an antiserum must be capable of precipitating the specific soluble substance of virulent strains. The authors succeeded in producing such a serum in horses by prolonged immunization with formalinized virulent influenza bacilli.

In addition they have pointed out that complement is necessary for the bactericidal action of a serum possessing the above-mentioned properties. Complement is practically always absent in the spinal fluid of patients with influenzal meningitis. Their treatment consists in the administration of antiserum plus fresh normal human serum as complement.

Therapeutic results so far have been encouraging. However, there appears to be early "walling off" in this disease and in many cases, even though it was possible to sterilize the spinal fluid, yet the patients would eventually die from localized lesions. In a personal communication from one of the authors (Fothergill) it has been learned that they have had ten complete cures to date. Because of early "walling off" it is needless to emphasize the necessity for early diagnosis.

These investigators have pointed out also certain important statistical data regarding the disease. It is primarily a disease of infancy, about 80 per cent of the cases occurring during the first two years of life. Furthermore, the disease is much more common than is generally supposed, for in their review of meningitis in the Infants' Hospital of Boston they found it the third most common type during the first two years of life. Tuberculous meningitis is the most frequent type with meningococcus meningitis second.

Recently Fothergill and Wright[9] have studied the possible reasons for the striking age incidence of influenzal meningitis, and have found the incidence to be accurately correlated with the bactericidal activity of whole blood from individuals of various age groups. Cord blood and blood during the first six weeks of life possess considerable bactericidal activity for virulent strains. This probably represents a passively acquired humoral antibody transferred from the mother through the placenta and is gradually lost during the first two months of life. It is interesting that the disease practically never occurs during the first two months of life. From two months to three years, the blood has practically no bactericidal activity and it is during this period that the majority of cases occur. From three years on, the killing power gradually rises. This killing power is probably due to an actively acquired immunity represented by a humoral antibody. This antibody, or its absence, is presumably important during the bacteremic stage of the disease.

TUBERCULOUS MENINGITIS

In 1932 Bokay[11] reported successful results in the treatment of five of seventeen cases of tuberculous meningitis (the diagnosis was established by both clinical and bacteriologic examinations) by energetic deep roentgen therapy. The author divided the cases into two classes, an exudative diffuse, and incurable group, and a local meningeal tuberculosis; it is this latter group that has responded to treatment. Wiener[12], encouraged by the report of Bokay, treated nine cases with roentgen therapy but was unable to obtain any improvement. In fact, it was his impression that the treatment hastened the death of the patients. However, some consideration must be given the fact that Wiener's cases were advanced while Bokay's were treated early in the course of their disease. Gordon[13] treated three cases by this method with the following results: "Two of them, according to the concepts presented, offered no favorable response to this method, in that the tuberculous infection of the meninges was only an accompanying part of a generalized miliary dissemination of the disease throughout the body. . . . The third patient was judged favorable for this form of treatment in that the infection of the meninges was the outstanding manifestation. . . . All three patients died, the first two rather promptly. The third one, the child being favorable for treatment, showed a well-marked clinical response early in treatment, with decreased fever, improvement of physical signs, and most important, improvement of the sensorium and general clinical appearance. A relapse occurred after six days with death on the eighth day." The extremely high mortality rate of this disease certainly justifies any therapeutic procedure which, in itself, is free from danger to the patient. Obviously further clinical investigation is not only warranted but indicated.

POLIOMYELITIS

In view of both the favorable and unfavorable discussion concerning the efficacy of convalescent serum in the treatment of acute preparalytic poliomyelitis, a brief review of this phase of the subject seems pertinent at this time of

the year. "The initial symptoms are much the same in all types of the disease and they may be quite as severe in the nonparalytic as in the paralytic." This statement by Neal[14] shows very clearly how difficult it is to determine the efficacy of any form of therapy instituted during the preparalytic period of acute poliomyelitis. Furthermore, it is impossible to evaluate properly the results of a given form of treatment unless the results can be compared with properly selected and supervised cases that did not receive treatment. The great difficulty in determining the value of convalescent serum (technic of obtaining and preparing serum[15]) has been the lack of control cases.

Soon after the introduction of this procedure in the treatment of acute preparalytic poliomyelitis, the literature contained favorable reports[16] and one received the impression that convalescent serum was of definite value. However, the only controls were those cases diagnosed after paralysis had appeared and these patients, in many instances, were not seen prior to that time. Obviously, herein lay an actual error in drawing conclusions, for the number of patients who recovered *without* paralysis and who were never seen, could not be included. In fact, the only conclusion that actually could be drawn from these early observations was that *if the serum is of value, it should be given early*[17] and before paralysis has developed. This same criticism, i.e., inadequate control groups, may be applied to the observations of recent authors[18, 19, 20, 21, 22, 23] who advocate the administration of convalescent serum. Andrews[24] has presented evidence to show that when a virus of the filtrable class has made its biologic union with the tissue cells it is no longer within reach of a neutralizing virus. This observation offers a possible explanation for the failure of the serum to be of value as a therapeutic agent.

Torian and Winters[25] are of the impression that convalescent serum is of little value in influencing the course of the disease, yet they treated only four cases during the preparalytic stage. Definite conclusions cannot be reached from such a small number of cases. In an extensive series of 927 preparalytic cases, 519 of which were treated with convalescent serum while the remaining 408 were not given serum, Park[26] found that paralysis developed in 19.6 per cent of the treated cases, in only 11 per cent of the untreated cases, and that the mortality rate among the former group was 3.8 per cent as compared with 0.9 per cent among the latter group. The recent observations of Kramer, Aycock, Solomon and Thenebe[27] in a carefully controlled series of treated and untreated cases also fail to give any statistical evidence of its value as a therapeutic agent.

In her conclusion on the treatment of poliomyelitis by convalescent serum Neal[28] states: "A group of more than 1000 cases, including those of Kramer and Aycock, diagnosed in the preparalytic stage, with approximately half treated by convalescent serum, shows nothing to indicate the therapeutic value of the serum, either in reducing case fatality or in preventing paralysis. Indeed, the slight difference between the treated and untreated groups is against the use of serum. Since the same results were shown by three groups of workers, the physicians of the Health Department and Hospitals, the physicians working under the direction of the subcommittee on Poliomyelitis of the Public Health Relations Committee of the New York Academy of Medicine, and Kramer and Aycock and their associates—it may be concluded that these cases were diagnosed and treated without personal bias. It is fair to say, however, that there is a belief that the percentage of moderately severe cases was a little greater in the treated children in the group studied under the direction of the Academy of Medicine." At the present time, further observations among carefully controlled cases would seem to be indicated before definite conclusions as to the specificity of action in preventing paralysis in this disease can be drawn.

Convalescent serum has been administered intrathecally, intravenously, and intramuscularly, either alone or in combination with one or more of the other methods. The intrathecal injection of the serum, though advocated by some, does not appear advisable, for it is invariably followed by an exaggeration of the clinical manifestations of meningeal irritation. Furthermore, it is a well-recognized fact that the presence of serum of any nature within the cerebrospinal fluid establishes a sterile meningitis and thereby possibly increases the already existing reaction. The intravenous route is preferable, for in this manner large amounts of the serum may be readily administered and rapidly distributed throughout the body. If convalescent serum cannot be obtained and it seems advantageous to administer this form of treatment, a blood transfusion may be employed. Certain authors[29] have advocated this procedure over serotherapy, as a donor can usually be obtained without delay and larger amounts can be given.

The administration of convalescent serum, or whole blood, as a widespread prophylactic procedure has been advocated, but at the present time the scientific evidence is insufficient to warrant its acceptance as a universal measure. In view of the fact that the majority of adults have acquired an immunity[30, 31] to the disease whereas young children have not, it may be advisable to administer serum intramuscularly or intravenously to this group when a member of the family has developed the disease. This passive immunity, however, is only temporary, lasting from five to seven weeks, and should the person be reëxposed after this period, a second injection of serum would have to be given. If such prophylactic measures were employed,

many children would unnecessarily be subjected to treatment simply because a case, or suspected case, occurred in their neighborhood.

NARCOLEPSY

In view of the increasing number of case reports in the literature and inasmuch as successful symptomatic treatment has recently been described by several writers, a brief review of this condition is of interest.

Narcolepsy, first described by Gelineau[32] in 1880, may be said to be characterized by two chief symptoms, i.e., an irresistible desire to sleep, and a feeling of generalized weakness following laughing or other emotional disturbance. The desire to sleep may occur at any time, regardless of the attending circumstances, and is unrelated to the amount of nocturnal rest; the patient may suddenly go to sleep while reading, eating, talking, walking, or working. This sleep resembles the "normal" sleep of an individual, although it may at times be accompanied by hallucinatory dreams which in themselves may cause the patient to awaken. As has been pointed out by Adie[33] there are occasions, however, when the weakness or cataplexy may result in closure of the eyes and inability to articulate, thereby giving the impression of sleep. The attacks of generalized weakness, or the cataplectic attacks, are precipitated by emotional disturbances, the most common of which is laughter. In fact, many of these patients refuse to laugh, knowing well that any such act will be immediately followed by a generalized sensation of weakness which may result in their collapse; less frequently fear, anger, or distress may produce this same sensation.

The etiology of this condition continues to be obscure, although there is good reason to believe that in certain cases it may be symptomatic, i.e., secondary to trauma, disturbances in glandular functions, infection, etc. Due to the fact that an increased number of these patients have been seen since 1918 (the year of the epidemics of influenza and encephalitis) it has been suggested that there might be a relationship between epidemic encephalitis and narcolepsy. Wilson[34], however, in his excellent review of the subject in 1928 makes the following statement: "Were one to argue from these considerations, a case might be made out for an actual increase in disordered conditions of sleep somehow related to the times in which we are living, and their ascription to processes out of epidemic encephalitis might be plausibly offered as one of explanation, but at present I am far indeed from being convinced of any frequent etiological association of the latter with the former." In regard to other etiological factors he states: "The data which have just been examined . . . are sufficient to show how diverse and heterogeneous is the etiology of the narcoleptic syndrome. To assort the causal factors is a matter of no little difficulty; a provisional arrangement might be made along the following lines:

1. Traumatic group.
2. Psychopathological group.
3. Endocrine group.
4. Epileptic group.
5. Toxic-infective group (epidemic encephalitis, etc.).
6. Circulatory group.
7. Pressure group (cerebral tumors, etc.).
8. Group in which no Grundkrankheit has been discovered."

Thrash[35], Spiller[36], Weech[37] and others have reported cases in which there appeared to be a definite relationship between endocrine dysfunction, encephalitis, and trauma and this disturbance.

The age incidence is such that narcolepsy is more frequently encountered in adults than in children. Cave[38] in a report of 42 cases found that the age of onset varied from nine to forty-six years with an average of twenty-five years in males and from eight to thirty-seven years in females, with an average of nineteen years. Levin[39], in a study of the published cases, found only 4 in which the age incidence was under ten years. In Cave's cases 34 were males and only 4 females; in Wilson's there were 37 males and 7 females, while in Adie's group of 20 (17 from the literature and 3 of his own) there were only 3 cases in females. Judging from the increased number of case reports of this condition in children during recent years one may later find that the disease is not so rare in this age group as the present statistics would lead us to believe.

The diagnosis can usually be established without difficulty if both symptoms are present. If the chief complaint is that of recurrent or frequent attacks of sleeping, there is reason for possible confusion before the proper diagnosis can be established. In some cases the cataplectic attacks may occur alone or for several months before the sleeping attacks appear, and it is in this group that one might be misled by considering epilepsy or petit mal. However, careful questioning will reveal the fact that the attacks of weakness occur only after *emotional* disturbances (especially laughing), are not accompanied by loss of consciousness (for these patients can be readily awakened), or by loss of sphincter control, and that they are not followed by stupor or prolonged periods of unnatural sleep.

The treatment of narcolepsy, like all idiopathic disturbances, has passed through many forms and processes and the result has always been the same. However, in 1931, Doyle and Daniels[40] reported successful symptomatic treatment by the oral administration of ephedrin sulphate. Quoting their summary: "Six pa-

tients suffering from narcolepsy were treated with ephedrin sulphate. Five were completely relieved of the essential symptoms of irresistible desire to sleep without apparent cause and cataplexy. The sixth patient is relieved of cataplexy and hypnagogic hallucinations, and somnolent tendencies are markedly improved." Similar results have subsequently been reported by Wahl[41], Collins[42], and Jacobsen[43]. The usual dose has been 3/8 of a grain, three times a day, although at times it has been necessary to increase this to 3/4 of a grain three times a day before satisfactory results could be obtained. Daniels'[44] recent monograph, with a detailed study of 377 cases, will prove of inestimable value to those interested in this subject.

PLACENTAL EXTRACT

One of the most outstanding advances in pediatrics has been the introduction and employment of human placental extract in both the control and modification of measles. McKhann and Chu[45, 46], in their original publications, demonstrated that placental protein extracts contained substances, presumably antibodies, which (1) neutralized diphtheria toxin, (2) blanched scarlet fever rashes, (3) neutralized poliomyelitis virus, and (4) prevented measles in exposed susceptible patients. At present the most extensive investigations in the use of this material have been in the prophylactic treatment of measles. Up to the present time it has been used in over seven hundred individuals[47] either for prevention or modification of the disease. In ninety-five non-immune patients exposed to measles in the course of hospitalization for other causes, the extract was administered early in the period of incubation in order to prevent the disease, and of this group ninety-one did not develop the disease while the remaining four developed very mild modified measles[48].

Among the factors which influence the effectiveness of the extract in the prophylaxis or modification of measles are (1) time of injection, (2) dosage and (3) potency of the material. By pooling human placentae in large lots to minimize the variation in antibody content of individual placentas, and by standardizing the dosage of the preparation on the basis of its protein content it has been possible to prepare an extract of almost uniform potency which promises to eliminate variability in results. For prevention of the disease a dosage of 5 cc. given within four days after exposure has been found to be effective whereas the same dosage given from the fourth to the ninth day after exposure has modified the disease. In certain instances large doses given after the appearance of Koplik's spots have altered the usual clinical course. Local reactions have occurred in about ten per cent of the treated patients and have been characterized by pain, tenderness and redness. At times this local reaction may be accompanied by malaise, headache. and a slight febrile reaction, all of which, however, usually subside within 24 to 36 hours. Although serum reactions have occurred in only two out of several hundred treated patients, yet this is sufficient to indicate that adrenalin should always be available whenever the extract is administered.

THE BACILLUS CALMETTE-GUERIN (BCG)

Recent developments concerning the inoculation of children against tuberculosis with the BCG vaccine may be considered under two general headings:

(1) The safety of the method. This question has been attacked, mainly by animal experimentation, to determine whether the organism preserved a fixed (and harmless) virulence under varying conditions. Petroff[49] has dissociated the bacillus on a gentian-violet egg medium into a predominant R and a rather infrequent S strain, which was pathogenic for animals. Sasano and Medlar[50] report a dissociation on culture on another medium, with a resultant strain definitely pathogenic for rabbits, guinea pigs, and calves. Feldman[51] has reported some evidence of pathogenicity of the bacillus for guinea pigs, but this was not heightened by subculture or animal passage. Dreyer and Vollum[52] have stated that the virulence of BCG vaccine could be enhanced by culturing in partial anaerobiasis.

On the other hand, there have been several reports by investigators who have failed to confirm the above findings, and who feel that BCG vaccine is non-pathogenic, at least for laboratory animals. Park and his co-workers[53] have repeated the procedures of Petroff, Sasano and Medlar, and Dreyer and Vollum, without any evidence of increased virulence. In three animals out of about 350 inoculated in these experiments a progressive tuberculosis developed, which the authors believed was due to cross-infection. Birkhaug[54] has repeated Petroff's and Dreyer and Vollum's work, without confirmation of their results. Griffith[55] reports a complete lack of virulence of the organism in guinea pigs, goats, and fowls, and a pneumonic disease in rabbits only after the intravenous injection of large doses.

(2) The production of immunity. It is interesting to note that although approximately a million children have received BCG vaccine yet there is no absolute proof or disproof of its value. The French statistics have not been adequately controlled[49]; in England the method has failed to gain any sanction[56]; and in Germany little inoculation has been reported since the

disaster at Lubeck in 1930. (The official investigation has decided that the deaths of vaccinated children at Lubeck were due to contamination or substitution of the culture used, rather than to change in virulence[57], although others who have also studied this matter have different views[49].) Perhaps the most valuable studies of the protective value of the vaccine in children are those of Wallgren[58] in Gothenburg, and of Park and his associates[53] in New York. In both of these investigations the best use of the BCG vaccine has been thought to be its intracutaneous administration to young infants in tuberculous families. Wallgren's report includes 194 children and Park's includes 150 children vaccinated in this manner. No deaths have occurred from tuberculosis in either group, while tuberculous mortality in suitable controls was about 3 per cent in each series. The protective value of BCG vaccine inoculation in animals has been repeatedly demonstrated; for a careful study the reader is referred to the work of Birkhaug[54].

It is possible, in a review of this kind, to mention only a very small portion of the literature on the general subject of the BCG vaccine. It is evident that large and extremely painstaking statistical studies, such as are developing in New York, will be necessary before definite proof of the advantages of BCG vaccination in children is obtainable.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASES 20221 AND 20222

DR. TRACY B. MALLORY: These next two cases are so similar that I am going to ask that both histories be read first and followed by a general discussion of both cases.

PRESENTATION OF CASE 20221

First admission. A white boy four and a half years of age was referred from another hospital. The complaints were sore throat and rapid heart.

Family history. His mother and father were well but were not living together. One sister two years of age was well. The second pregnancy resulted in a miscarriage at four months. No family history was obtained of allergic manifestations, tuberculosis, lues, insanity, kidney or heart disease in any form.

Past history. The patient was delivered normally at full term and was active and vigorous. He was breast fed for nine months. Cod liver oil and orange juice were started at three months, vegetables and cereals at six months. His development was normal. He had possible measles at three months, with rash, conjunctivitis and bronchitis, chicken pox at three and a half years, and scarlet fever at four years, with no sequelae noted. He had never had chorea or joint trouble, but did have frequent sore throats and colds.

Present illness. Two and a half years before admission, at two years of age, he had his first attack of tachycardia. His mother found him lying on the ground in the yard, but not unconscious. He complained of pain over the heart. Although he did not appear to be very ill, he was put to bed. Shortly afterward he began to vomit. For about twelve hours he wished to be let alone, and lay quietly. Then the attack passed off. Attacks like this recurred about once a month. One attack with severe vomiting for three days culminated in unconsciousness, and he was taken to a hospital. Since that time he had had eleven admissions for the same cause. His mother felt that his attacks increased in frequency following a fright, and she believed that fear brought them on. They were however preceded by a severe tonsillitis. They lasted from twenty minutes to two weeks. During the six weeks he was in bed with scarlet fever some six months before admission he had no attacks. Lately attacks had occurred as often as once a week, but were less severe. The boy could usually tell when an attack was coming on. He became fretful and very irritable. During this time his pulse would not be rapid. He might go to bed voluntarily before the actual onset of the attack. During the attacks he vomited and refused all food. His tongue became coated. The stools were normal in number but showed undigested food. If the attacks lasted several days his skin became blotchy; there was circumoral pallor and later general pallor. If the attacks lasted a week his ankles, hands and face began to swell. Medication had not affected the attacks.

Physical examination showed a well developed and nourished, somewhat pale, coöperative boy. His tonsils were moderately injected and cryptic, and there were yellowish spots on the pharynx and tonsils. There was moderate cervical adenopathy. The heart rate was 90, the respiration 24, and the temperature 101°. The blood pressure was 92/52. The left border of dullness was 6.5 centimeters from the midsternal line, almost a centimeter beyond the midclavicular line. There was a soft systolic murmur heard loudest in the fifth interspace, also heard at the apex and in the pulmonic area. There was an occasional extrasystole, and a questionable third sound along the sternal border at the fourth left interspace.

Laboratory work. The examination of the urine was negative. Tuberculin and Hinton tests were negative. The stools were negative. Examination of the blood showed a red cell count of 4,300,000 with a hemoglobin of 65 per cent and a white cell count ranging between 17,000 and 35,000, with 70 per cent polymorphonuclears.

An electrocardiogram on admission showed sinus arrhythmia, rate 50 to 70, prominent P_1 and P_2, left axis deviation, and slightly slurred and widened QRS. During an attack an electrocardiogram showed paroxysmal auricular tachycardia, rate 280.

Course. The child had several attacks of paroxysmal tachycardia lasting four to eight days, the rate going as high as 280 per minute. The attacks were as described in the history. The apical rate during the attacks ranged between 200 and 280, with a radial pulse of 100 to 150. Between the attacks the rhythm was often quite irregular, with a rate as low as 60 associated with coupled beats. Acetyl cholin was given during one attack without definite effect. Injection of the left dorsal sympathetic ganglia with novocain did not affect the tachycardia. Injection of the right dorsal ganglia with novocain stopped the paroxysm immediately. On two subsequent occasions a paroxysm was stopped by injection of novocain. In order that the effect might be more permanent, on the last occasion after the novocain was injected alcohol was injected into the ganglia; no further paroxysm occurred dur-

ing the four days he remained in the hospital.

At the end of five and a half weeks the child was discharged.

History of interval. He was well until two weeks after his discharge, when an attack started which lasted until half past three p.m. on the fourteenth day after its onset, at which time the heart resumed its normal rhythm. Later in the day, after he had begun to vomit again and his pulse rate had increased, he was brought into the hospital.

Second admission, a month after his previous discharge.

He was pale and in some distress. He had definitely enlarged cervical glands. The heart was not enlarged; no murmurs were heard; the rate was 220. The liver was not tender, but was two and a half fingerbreadths below the costal margin. The blood pressure was 120/80. Physical examination was otherwise negative. The laboratory findings were negative except for a white blood cell count of 25,400.

Course. The pulse remained high. The liver increased slightly in size. There was no edema. On the fifth day in the hospital the right inferior cervical and upper dorsal ganglia were injected with novocain. Within one hour the heart rate dropped from 240 to 100 beats per minute. On the following day the child was brighter and much more active. His color was good. The liver was only one and a half fingerbreadths below the costal margin. The pulse was slow and regular.

At four o'clock in the morning of the second day following the injection, the child sat up in bed and screamed with pain. He then became semicomatose. His pulse rate increased. Response was difficult to obtain. At eight o'clock, although acutely ill, he could understand questions. Both pupils reacted to light and were equal in size. There was a drooping of the left eyelid and left facial paralysis. The tongue deviated to the left and a left hemiplegia was present. The right arm and hand were dry and cold and the left arm warm and moist. At ten a.m. the pulse was 100. There were nystagmoid motions of the eyes and twitching of parts of the body. The abdomen became distended. The child died at half past nine p.m., after expelling mucus and blood tinged froth from his mouth.

Presentation of Case 20222

A twenty months old white native born male infant entered the hospital with the complaint of cough and occasional vomiting since an attack of pertussis two months earlier.

The previous summer a doctor had said he was anemic, but he seemed otherwise well until August, when he had whooping cough. After three weeks this progressed into pneumonia, and he spent the next month in a hospital for contagious diseases. Upon returning home from that hospital he still had some cough, mostly at night, and some of the attacks were followed by vomiting. There was no temperature. During the three weeks before admission he had been restless, irritable and fretful and his appetite had been poor.

The past history showed a full term normal delivery. The weight was eight pounds, and he was considered normal in every way at birth. From the first he received a formula of condensed milk and water, with no cod liver oil or orange juice. At entry he was still being fed on a formula, with vegetables and egg yolk. He sat up at four months, but had not yet walked at twenty months. His general development had been slow. He was very sick with bronchopneumonia at nine months of age.

The mother and father were aged nineteen and twenty-two respectively, and were in good health. One sibling died at six months with whooping cough. The family history was otherwise negative.

Physical examination showed a pale, chubby, washed-out looking infant not acutely ill. There were signs of rickets in prominent frontal bosses and a rosary. There was some mucoid discharge from the nose. The throat was reddened, and there were small shotty glands palpable over the neck. A few scattered musical râles were heard over the chest. The heart was enlarged somewhat to the left of the midclavicular line. The rate was 160 and regular. No murmurs were heard. The abdomen was a "pot belly" with a prominent umbilical hernia. The liver edge was palpable at the level of the iliac crest.

The temperature was 101°. The respirations were 30.

Laboratory work. The blood showed a red cell count of 5,800,000, a white cell count of 25,100 and 72 per cent hemoglobin. The blood smear showed a normal differential count, with marked polychromatophilia and anisocytosis of the red cells. Hinton and tuberculin tests were negative.

X-ray examination of the chest showed the hilus shadows prominent on both sides, pleural thickening on both sides, and a prominent bulge in the region of the right auricle.

An electrocardiogram showed paroxysmal tachycardia with a rate of 200.

For the first two days the pulse varied between 110 and 180. On two occasions during the first ten days in the hospital Dr. J. C. White injected the first and second right thoracic sympathetic ganglia and on one occasion the left with novocain, producing a slight Horner's syndrome but no appreciable effect on the pulse. Following this the child was digitalized in an endeavor to slow the pulse; but there was no

slowing of the rate, although the child's general condition was better. Digitalis was then discontinued, and a tincture of veratrin, one minim three times daily, was begun. For a time the boy seemed to be doing well, the heart showing many dropped beats, but clinically he was not so well as he had been under digitalis. He was next given quinidine sulphate, the dose starting with two and a half grains daily and slowly increasing. He did not do well under this régime, and when the dosage reached fifteen grains daily he went into collapse, but recovered when stimulants were given. The quinidine was stopped. Acetyl cholin was tried once, with only slight physiological effect and no effect on the pulse. The patient was next digitalized and put on quinidine sulphate one half grain three times daily by rectum. On this régime the pulse steadily tended downward, and after five days the electrocardiogram showed a normal rhythm for the first time since he entered the hospital, with a rate of 140. After stopping digitalis and quinidine for forty-eight hours the normal rhythm became interrupted by many auricular beats, which disappeared upon restoring quinidine but omitting digitalis. Soon afterward he developed pneumonia and otitis media, but recovered. During the pneumonia his tachycardia returned, and resisted all attempts to restore normal rhythm with digitalis and quinidine. On recovery from the pneumonia, the child again showed improvement in general condition and at times a normal pulse rate (120 per minute). At other times the pulse varied up to 200. He developed pneumonia (left lower lobe) again five weeks after recovery from the previous infection. He was very ill; the pulse rose to between 200 and 220. The temperature then rose steadily to 107°, and he died three days after pulmonary signs appeared, five months after he entered the hospital.

X-ray Interpretation

Dr. Aubrey O. Hampton: This film was taken twenty-two days before death and this one a month and a half. The total transverse measurement of the heart is 9.6 centimeters. The internal diameter of the chest is 17.8 centimeters. I think we cannot compare these two because in this case there was rotation. The diaphragm has come down after the second examination and there seems to be more passive congestion in the lung. The enlargement of the heart is diffuse, but seems to be more to the left in this film, and here we have a triangular shaped heart, but I think that is due to the position of the patient.

Clinical Discussion

Dr. Mallory: I believe you saw these cases, Dr. Eustis. Would you like to say a word?

Dr. Richard S. Eustis: I saw the second of the two cases at the end, after the child's first attack of pneumonia, while he was running apparently a paroxysmal tachycardia. We did not know what else to call it. We tried to control it with quinidine without much effect. Dr. Boone knows the details of that far better than I do.

Clinically he was a cheerful child sitting up in bed smiling and sleeping most of the time, with a pulse rate of 200. I suspect that the pneumonia was a terminal complication and not the real cause of death, because his pulse rate started climbing before his temperature went up and signs of pneumonia appeared. I suspect that his heart began to go bad and he then developed a pneumonia and died of pneumonia.

I am completely at sea as to the underlying cause of such a condition. I have never seen anything like it before. I have heard of comparatively innocuous paroxysmal tachycardia in older children and young adults, but this picture is entirely novel to me. I hope Dr. Mallory can tell us more about it.

Dr. Joseph Garland: I saw only the second of these two children when he first came in. I think both the cases illustrate the difficulty encountered in controlling paroxysmal tachycardia.

Dr. Harold L. Higgins: I think in the second case the question of pertussis playing a rôle in the etiology comes up. I looked in many textbooks regarding pertussis, and I find no mention of pertussis affecting the heart at all, except that following severe paroxysms of coughing there is sometimes dilatation of the right ventricle. This child had pertussis and a bronchopneumonia which went with it and kept recurring, and no doubt this played some rôle in his cardiac condition.

As to the first case, the cause of death was pretty clearly cerebral embolus. This child showed a much more rapid tachycardia than the second child. He would show distinct signs of cardiac failure after his paroxysmal tachycardia had continued over a period of twenty-four hours. The second child seemed to stand the tachycardia quite well until he got an infection. When his bronchopneumonia became marked we found signs of cardiac failure.

Dr. Paul D. White: These are two rather extraordinary cases of course, not showing the usual results of paroxysmal tachycardia, a condition that is ordinarily considered to be unimportant, well limited in attacks and allowing many years of life. There are patients on record who have had paroxysms of tachycardia for thirty or forty years without causing any serious heart involvement and without any heart disease to start with. The chances of the presence of heart disease in a patient who has par-

oxysmal tachycardia are probably only one in three or four, perhaps less. Serious paroxysms are very uncommon. Here, however, we have two very young children who had fatal complications. There was no clear proof during their illness of organic heart disease, although there was some indication of cardiac enlargement in the second case and in the first child there was an abnormality of the electrocardiogram other than the paroxysmal tachycardia; when the rhythm returned to normal the electrocardiogram revealed abnormal QRS waves, which ordinarily indicate intraventricular block and lead us to think of myocardial disease. We have not found paroxysmal tachycardia, however, indicative of congenital defects or in any of the rheumatic involvements of the heart with which we are ordinarily familiar.

I will pass around these records. The first set is a group of three electrocardiograms taken from the first child. The first record of the set was obtained during a paroxysm of tachycardia with a heart rate of 230 and some intraventricular block. The second and third records were taken during the injection of novocain and show either normal rhythm or an alternation of normal rhythm and short paroxysms of tachycardia; there is a persistence of the intraventricular block during the normal rhythm. The second set of records, from the second child, shows normal QRS waves with inverted P waves of auricular paroxysmal tachycardia; in the last strip of the first record you will note that there is one dropped beat, due to the production or occurrence of block. We were able by the use of digitalis to increase the degree of this block and therefore to reduce the heart rate. It is possible that, if there had not been pneumonia, digitalis and quinidine might eventually have controlled the situation. That of course remains in doubt.

The very fast heart rate during the paroxysms of tachycardia in these two children is rather characteristic of this disturbance of rhythm in extreme youth. The fastest rates that I know of were in two infants, one a case of Dr. Paul Emerson with a rate of 310 and the other a recent case of Dr. Lyons of Washington with a rate of 320, both proved electrocardiographically; the latter case so far as I know is the world's record. The fastest heart (ventricular) rate that we have recorded electrocardiographically in our laboratory at the Massachusetts General Hospital in an adult was 273; this case, a woman, recovered and lived for years after that record was obtained.

DR. HOWARD B. SPRAGUE: I had charge of the first case when the child was at the other hospital. We were impressed by the fact that change in position or emotional stimuli would produce or terminate the attacks. Standing him on his head would stop an attack. On the other hand falling out of bed might start one. It is very difficult for me to see how such procedures alter the condition of the myocardium. One would suppose that the effect was produced through some nervous reflex.

The second case I saw also and at one time brought up with Dr. James C. White the question of cutting down upon and electrically stimulating the vagus nerves. I hope Dr. White will tell us something about that procedure.

DR. T. DUCKETT JONES: The first case I saw over a period of two years at the two outside hospitals. During one attack they found the child had a blood sugar of fifty and gave him intravenous glucose and the paroxysm suddenly stopped. With subsequent attacks however the blood sugar was always normal, so the influence of hypoglycemia did not help. The emotional side in this case was very striking. One day I went into the ward and picked up a balloon that was lying on the table and the child became violently angry and started to have a paroxysm of tachycardia at about 200 a minute. I hope Dr. White is going to have something illuminating to say on that. So far as I know everything was tried for a period of two years that possibly could have been tried. That is why he was sent down to Dr. White.

DR. JAMES C. WHITE: Of course paroxysmal tachycardia is supposed to be an intrinsic disease of the pace regulating mechanism of the right auricle. It was a question whether doing anything to reduce the activity of the extrinsic cardiac nerves could control these attacks by diminishing the irritability of the sino-auricular node. There was some indication for that in this patient's history, as his attacks were often set off by emotional factors and also the fact that when he had a high fever his attacks stopped. A high fever we know diminishes certain forms of sympathetic activity. In order to test the effect of paralysis of the cardiac accelerator nerves, I blocked the upper four thoracic sympathetic ganglia on the left side with novocain. This had no effect at all. When I tried it on the right side there was an immediate cessation of his tachycardia that you can see by looking at the electrocardiograms. It was successful twice.

I think the mistake we made with him was in not going on that evidence and resecting these ganglia rather than attempting an alcoholic injection, with which I had had no experience in so small a child. If operation had been done there is a fairly good chance that he would not have developed any more paroxysmal tachycardia. Possibly if that had been done in the beginning he would not have shot off his final and fatal embolus. Dr. Leriche in Strasbourg has operated on two of these cases. One has been followed for a period of a great many years with no recurrence of the tachycardia. I believe Dr. Paul White thinks that the first case of angina pectoris that was operated on in 1916 by

Jonnesco was very probably paroxysmal tachycardia.

In the second case novocain gave no evidence of benefit. Instead of cutting down and stimulating the vagus, as Dr. Sprague suggested, he was given acetyl cholin, which is known to produce a very definite stimulation of vagal activity. This gave no benefit, and further neurosurgical investigation was discontinued.

Clinical Diagnoses, Case 20221

Paroxysmal auricular tachycardia.
Cerebral embolism.
Pulmonary congestion.
Left hemiplegia.

Anatomic Diagnoses, Case 20221

(Paroxysmal auricular tachycardia.)
Cardiac hypertrophy.
Mural thrombus, left ventricular wall.
Cerebral embolism and infarction.
Congenital bicuspid aortic valve.
Endocarditis, chronic, tricuspid.
Diffuse thickening of the endocardium.
Chronic passive congestion of liver and spleen.
Hydrothorax, bilateral.
Hydropericardium.
Pulmonary congestion.
Dilatation of stomach.
Intussusception.

Clinical Diagnoses, Case 20222

Paroxysmal tachycardia.
Lobar pneumonia, left lower.

Dr. Harold L. Higgins' Diagnoses, Case 20222

Paroxysmal tachycardia.
Bronchopneumonia, left lower.

Anatomic Diagnoses, Case 20222

(Paroxysmal auricular tachycardia.)
Hypertrophy and dilatation of heart, especially right auricle.
Bronchopneumonia, bilateral.
Petechial hemorrhages of mesentery, thymus, epicardium.
Chronic fibrous pleuritis, left.
Hydrothorax, left, slight.

Pathologic Discussion

Dr. Mallory: The autopsies on these two children showed nothing anatomical which might explain their tachycardia.

The older child, the first case, had an abnormal heart shown for one thing by a congenital bicuspid valve. There was absolutely no evidence of endocarditis of this valve and the microscopic anatomy fits perfectly the criteria of Lewis and Grant.* We found moreover in the apex of the left ventricle a rather loosely adherent antemortem thrombus. It was attached at only one very minute spot to the ventricular wall and the ventricular musculature immediately under it was perfectly normal. I think there is no doubt that it was from this thrombus that the embolus broke off which lodged in one of his anterior cerebral arteries in the frontal lobe and was the immediate cause of death.

*Lewis, Thomas and Grant, Ronald T.: Heart 10: 21, 1922.

A point which immediately arrested our attention was the unusual thickness of the endocardium of the ventricles, particularly the left one. When a heart is opened at the autopsy table the endocardium of the ventricles is very translucent and one can easily see the pink musculature through it. The endocardium is normally of varying thickness in the individual chambers and in the left auricle is normally so opaque that it appears as a white layer through which we cannot see the musculature at all. In this child the endocardium throughout the heart was of the same thick white opaque character that you normally find in the left auricle. This was particularly striking in the left ventricle. Two minute fibrous tabs, presumably healed vegetations, were found on the tricuspid valve.

Microscopic sections show a few very minute areas of scarring and fibrosis within the myocardium. The endocardium shows collagenous and elastic thickening of extreme degree. I have never seen, except over an old area of infarction in an elderly man, so thick an endocardium as this boy shows. Otherwise the findings are entirely negative. It is possible to imagine that this thickened endocardium might have interfered in some way with conduction in the bundle. Heart block, however, was an inconspicuous feature of the case. The sino-auricular node is the only point where an anatomic lesion would explain the auricular tachycardia, and the node so far as we can make out is entirely negative.

In the second case there was a slight degree of thickening of the endocardium, although not nearly so marked as this, and numerous sections from all parts of the heart, including the sino-auricular node, fail to show any evidence of myocardial scarring or any acute myocardial process.

It seems to me that one must consider these cases as essentially functional heart disease and has to admit the possibility of death from functional disease. In living patients in the clinics we are always constantly aware of the frequency of functional disease, but on the autopsy table one ordinarily assumes that it can be ruled out. I do not believe that we are always justified in doing so, however. I feel quite sure that there are certain types of functional disease so severe that they can cause death, and I personally should be inclined to place these two cases in the group.

Dr. Paul D. White: May I add a word

about the first case? I should think that if there is that extensive abnormality of the endocardium it must also have involved the right auricle. There might thus be an organic basis for irritability of any part of the auricular myocardium which would render more likely the precipitation of paroxysmal tachycardia through nervous channels. It is conceivable that this abnormality of the endocardium might involve also the branches of the auriculoventricular bundle of His which lie just under and in contact with the endocardium, thus explaining the intraventricular block found in the electrocardiogram. We have seen such thickening of the endocardium in a few instances of congenital heart disease, particularly in two or three in which there was pulmonary stenosis or stenosis of the infundibulum of the right ventricle, which suggests that there might have been an intrauterine infection, that is, an endocardial infection during fetal life with or without involvement of the valves.

DR. MALLORY: I think one is constantly meeting the problem as to which is cart and which is horse. I do not think we can settle it in this case.

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THE WORCESTER MEETING

LAST June the Worcester District Medical Society cordially invited the Massachusetts Medical Society to hold its next annual meeting in Worcester. Often such an invitation is purely perfunctory, and those extending it feel that their responsibility ends with its acceptance. But the Fellows of the Worcester District feel keenly their position as hosts, and have done everything in their power to make the meeting a success. At their annual meeting on May 9, 1934 they unanimously voted to assume the expense of entertainment for the Fellows and their wives, and a very generous sum was set aside for that purpose.

The Local Committee of Arrangements has been most coöperative, and helpful to the State Committee, and has planned excellent clinics for Monday, June 4, at all the leading hospitals of the city. After these clinics, luncheons will be served at the hospitals through the courtesy of the Worcester District Medical Society. The Local Committee has also arranged with the Worcester, Tatnuck, and Wachusett Country Clubs to permit all Fellows and guests of the Massachusetts Medical Society to play golf as guests of the Worcester District Medical Society at any time during the three days of the meeting.

The Annual Meetings have always provided for the Fellows of the Massachusetts Medical Society an opportunity not only to refresh or make additions to their professional knowledge, but also to renew old friendships and make new ones. The value of the social aspect of these meetings should not be underestimated. In the past when entertainment has been provided for ladies, many Fellows have come with their wives, and the enjoyment of the meetings has been greatly enhanced.

Our hosts of this year have arranged an elaborate program of entertainment for visiting ladies, the details of which appear on page 1194 of this issue.

Women are taking an increasing interest in the activities of the medical profession. Should we not now consider the advisability of forming in Massachusetts a branch of the Woman's Auxiliary to the American Medical Association?

It is a pleasure for us to welcome the wives of Fellows at our meetings, and we feel that such contacts should give them a better understanding of their husbands' work.

"CANCER IS CURABLE"

THE bright side of the cancer subject needs to be occasionally repolished and presented anew. The results of all the work that is going steadily on merit a feeling of optimism, but there is enough that is disheartening to make us occasionally forget that progress is being made. It is well, therefore, that the *Cancer Clinic Bulletin* of the Massachusetts Department of Public Health should this month summarize the symposium conducted by the American College of Surgeons in October on the curability of cancer.

Six objectives were in view when this symposium was proposed:

1. To impress upon the practitioners of scientific medicine, and indirectly upon the public, the fact that carcinoma is curable by the use of well-known and established methods of treatment.

2. To point out in a convincing manner that if all cases of cancer could be diagnosed early and treated promptly in their incipiency, the annual death rate from the disease, now recorded as one hundred and fifty thousand in the

United States and Canada, would be reduced by at least thirty-three per cent, or fifty thousand per year.

3. To bring together a group of distinguished clinicians to present definite statements of the impressive number of cases of cancer that have actually been cured.

4. To secure the maximum of ethical publicity of the reports.

5. To convince the profession and the public that even though cancer is already apparently in a later stage of its development, if it is subjected to proper treatment its progress may often be delayed and the disease not infrequently cured.

6. To establish a universal policy among physicians and surgeons to report cancer cures rather than to present the involved comparative statistics that dwell particularly on the cases not cured.

Reported cancer cures during the years 1932 and 1933 are forthwith summarized, the grand total of five year and over cures amounting to twenty-four thousand, four hundred and forty-eight. The percentage of cures, of course, varies in different localities and in different types of malignancy.

Dr. Malcolm B. Clopton of St. Louis reported one hundred and twenty-six breast cancers. Of thirty-seven cases with the disease in the breast alone, there were twenty-four, or sixty-four per cent living and well five years after operation. Of eighty-nine cases in which the cancer extended beyond the breast, twenty-seven, or thirty per cent were alive and well. Harrington and Judd, of Rochester, Minnesota, reported on a series of two thousand six hundred and fifty-six cases that twenty-six per cent of those with, and seventy-one per cent of those without involvement of lymph nodes had survived the orthodox five-year period. Greenough of Boston reported a total salvage of twenty per cent of cases operated on between the years 1894 and 1926. Other similarly encouraging reports were included.

Of one hundred and fourteen cases with carcinoma of the cervix Anspach of Philadelphia reported 18.4 per cent cures, and Crossen of St. Louis twenty-four per cent. Healy of New York reported eighty-five per cent of five-year cures in cancer of the body of the uterus. Charles C. Lund of Boston presented an analysis of the cases of carcinoma of the buccal cavity and lips treated at the Collis P. Huntington Memorial Hospital from 1918 to 1926, reporting that of four hundred and thirty-two cases of carcinoma of the lip and one thousand one hundred and thirteen cases of carcinoma of the buccal mucosa, one hundred and seventy-five and sixty-two, respectively, were cured.

An imposing array of figures, they prove that the case against cancer is by no means hopeless, indicating at the same time that as progress in early diagnosis and effective treatment is made, still greater improvement will result.

AN INTERESTING SITUATION IN MICHIGAN

At a meeting of the House of Delegates of the Michigan State Medical Society, April 12, 1934, approval of the principles of Health Insurance was voted and the Committee on Medical Economics of this body was instructed to proceed in developing a practical plan relating thereto.

In the history of this movement certain facts and details of procedure are of interest.

In July, 1933, the Committee on Medical Economics of the Michigan Society was directed "to continue its study, and prepare and present plans for Health Insurance". At this time the House of Delegates adopted the following designated policies for the guidance of the Committee:

1. Free choice of physician by the insured.
2. Limitation of benefits to those of medical service.
3. The control of medical service benefits by the profession.
4. The exclusion of individuals or organizations that might engage in health insurance for profit.

After a meeting in September, 1933, the Committee undertook as its first task this matter of health insurance, and since conflicting reports concerning the validity of the principles of health insurance gave the Committee concern, it was felt that a field study of health insurance in Europe should be conducted. In anticipation of this study the chairman of the Executive Committee of the Michigan Society House of Delegates felt that there should be a meeting in Chicago with representatives of the American Medical Association. This conference was held and is described by Dr. Henry A. Luce, speaker of the Michigan House of Delegates in the following words:

"The Chairman of the Executive Committee concluded that the subject was one that concerned not only Michigan but all the other states as well. He, therefore, concluded that the best results would be obtained if the Executive Committee met in Chicago with our employees in the national organization. The purpose of such a meeting was obvious. Michigan sought advice and guidance in the solution of its problems.

"To me the results of this meeting with our employees were both unsatisfactory and disturbing. Those present, in addition to the Executive Committee and Dr. Bruce, were Dr.

West, Dr. Leland, Dr. Woodward and Dr. Carey, Ex-President of the A. M. A.

"The Michigan delegation placed its problem before these men and asked for specific information.

"The information requested was not forthcoming and the general attitude seemed antagonistic. While Dr. West kindly explained the workings of the A. M. A., the discourse was not on the subject and failed to answer our questions. Dr. Leland appeared to be guarding 107 pages of manuscript on the subject of health insurance, but stated he was not in a position to report. Dr. Carey magnanimously offered advice that seemed to be a bit gratuitous. He said that we should thoroughly thrash out the whole subject, that we should not involve ourselves, and that Michigan should delay lest it get into trouble. In short, the advice was 'Do nothing.' All this, of course, gave little information and less comfort to the Michigan Executive Committee. During the discussion, Dr. Bruce asked Dr. West certain questions concerning the publications of the A. M. A., and the answers were anything but satisfactory. The representative of the A. M. A. assured the Executive Committee of the Michigan State Medical Society that they had no objection to Michigan making an independent study of European plans."

Six days later Dr. Henry A. Luce and Dr. Nathan Sinai went abroad for a study of Health Insurance, and on their return submitted a report to the Economics Committee of the House of Delegates of the Michigan Society, February 23, 1934, and a copy was forwarded to the Executive Committee of the Board of Trustees of the A. M. A.

A copy of this report appears in the supplement of the *Journal of the Michigan State Medical Society* for May, 1934. This report should be carefully read by all interested in health insurance, because it sets forth the evolution of the present plan in operation in England.

On receipt of this Luce-Sinai report the Economics Committee prepared a plan with recommendations under the title of "A Plan for Mutual Health Service" which was submitted to the House of Delegates of the State Society, at a special session, April 12, 1934, and signed by the several members of the Committee with Dr. Warnshuis as secretary.

This report, a copy of which appears on page 1185 of this issue, of the *New England Journal of Medicine*, after due consideration and prolonged debate was adopted by the House of Delegates by a vote of sixty-two to nine. We hope that it will be studied by doctors and dentists. An interesting feature of this movement is the coöperation of the dentists with the physicians as shown by the financing of the Luce-Sinai Commission by a joint grant from the Michigan State Medical Society and the American College of Dentists. This is evidence of the growing appreciation of the important part which dentists play in health problems.

After the acceptance of the report of the Economics Committee, the House of Delegates adopted a resolution directing this committee to prepare and present a budget for completing the study of the program outlined in the report and make plans for carrying the recommendations into operation.

Thus far the Michigan movement is apparently waiting to learn of any reaction of the House of Delegates of the American Medical Association to the report which was forwarded to the officers of that body, and the creating of a budget for trials of the suggested plans in some of the counties of Michigan.

Here is evidence of concentration of constructive medical interest in some phases of so-called health insurance under the direction of a state medical society.

It seems strange that organized medicine in this country has been loath to take the initiative in adopting some plan which, under medical guidance, might serve the interests of certain groups in their health problems. A definite obstructive attitude has been evident on many occasions with bitter and uncompromising criticism. The history of health insurance in other countries has placed the medical profession in an unfortunate position from which it has been only partly extricated.

Michigan has made an effort to build a constructive program based on the recognition of her responsibility to meet local conditions.

The reaction of the House of Delegates of the American Medical Association to this movement of one of its constituent societies will be watched with interest. Since the Michigan Society has taken steps to have this plan considered by the legislative and executive body of the A. M. A., the delegates of other state societies ought to be familiar with the plan presented by the Michigan Society when it comes up for discussion in the meeting of the House of Delegates of the A. M. A.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MUNRO, DONALD. A.B., M.D. Harvard University Medical School 1916. F.A.C.S. Assistant Professor of Neurological Surgery, Harvard Medical School. Visiting Surgeon, Neurological Surgery, Boston City Hospital. His subject is "The Diagnosis and Treatment of Subdural Hematomata. A Report of Sixty-two Cases." Page 1145. Address: Boston City Hospital, 918 Medical Building, Boston, Massachusetts.

PATTERSON, ROBERT L. M.D. Harvard University Medical School 1932. Interne, Surgical Service, Peter Bent Brigham Hospital. Address: Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Massachusetts. Associated with him is

GROSS, ROBERT E. M.D. Harvard University Medical School 1931. Interne, Pathological Service, Peter Bent Brigham Hospital. Address: Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Massachusetts. Their subject is "Adenocarcinoma of the Stomach." Page 1161.

ROWE, ALLAN WINTER. S.B., S.M., A.M., Ph.D. Director of Research, Evans Memorial, Massachusetts Memorial Hospitals. Professor of Chemistry, Boston University School of Medicine. Address: 80 East Concord Street, Boston, Massachusetts. Associated with him is

MCMANUS, MARY A. A.B. Assistant in Biochemistry, Evans Memorial, Massachusetts Memorial Hospitals. Address: 80 East Concord Street, Boston, Massachusetts. And

PLUMMER, ALBERT J. A.B., A.M. Fellow in Biochemistry, Evans Memorial, Massachusetts Memorial Hospitals. Address: 80 East Concord Street, Boston, Massachusetts. Their subject is "The Metabolism of Levulose: IV. The Hepatic Influence on the Utilization of Galactose and Levulose." Page 1163.

COTTON, FREDERIC JAY. M.D., and MORRISON, GORDON MACKAY, M.D. See page 817, issue of April 12, for records of authors. Their subject is "Pathological Dislocations of the Shoulder Backward and Rotation Deformity." Page 1169.

ELEY, R. CANNON. M.D. University of Virginia Department of Medicine 1925. Associate Physician, Infants' Hospital and Children's Hospital, Boston. Instructor in Pediatrics and Communicable Diseases, Harvard Medical School. His subject is "Advances in Pediatrics." Page 1170. Address: 264 Beacon Street, Boston, Massachusetts.

The Massachusetts Medical Society

ANNUAL MEETING OF THE COUNCIL

THE annual meeting of the Council will be held in the Ballroom of the Hotel Bancroft, Worcester, on Tuesday, June 5, 1934, at 12 o'clock, noon.

Business:

1. Reading record of last meeting in abstract.
2. Nominating Committee retire to deliberate.
3. Report of Committee on Membership and Finance.
4. Reports of committees to consider petitions for restoration to the privileges of fellowship and new committees to be appointed.
5. Report of the Treasurer.
6. Action on draft of revised By-Laws, submitted to each Councilor with the official program, May 6. A second revision by the committee appointed by the Council.
7. Reports of Standing Committees and Special Committees.
8. Election of Officers and Orator by ballot.
9. Appointment of committees for ensuing year, both Standing and Special.
10. Incidental business.

WALTER L. BURRAGE, *Secretary.*
Brookline, May 29, 1934.

Councilors are asked to sign one of the two attendance books before the meeting.

MISCELLANY

REPORT OF THE COMMITTEE ON MEDICAL ECONOMICS TO THE HOUSE OF DELEGATES OF THE MICHIGAN STATE MEDICAL SOCIETY†

MUTUAL HEALTH SERVICE

1. The purpose of Mutual Health Service is to provide, through the agency of a non-profit organization, health services at agreed costs to employed persons and the families of employed persons whose annual income does not exceed $.....*

For the purposes of this plan the word "family" is defined as including, in addition to the employee, any or all of the following *unemployed and dependent members living in the home of the employee:* wife (husband), sons, daughters.

With the exception of such limitations as are hereinafter provided, the term "health services" shall include the services of Doctors of Medicine, Doctors of Dentistry, Registered Nurses, Pharmacists, laboratories and hospitals as well as drugs and medical, surgical and optical appliances.

2. In accordance with the spirit of mutual responsibility between those who provide and those who benefit by the provision of health services the general direction of Mutual Health Service shall be as follows:

A. Board of Governors.

The Board of Governors, the members of which shall serve without pay, shall be constituted as follows:

Three (3) Doctors of Medicine elected by the House of Delegates of the Michigan State Medical

†Supplement to Journal Michigan State Medical Society, May, 1934, Vol. XXXIII, No. 5.

*The upper income limit was placed at $1500.

Society to serve for a period of three years, except that of the first three elected one shall serve for one year, one for two years and one for three years. Thereafter one Doctor of Medicine shall be elected annually for a period of three years.

One (1) Doctor of Dental Surgery elected by the House of Delegates of the Michigan State Dental Society to serve for a period of three years.

One (1) Pharmacist elected by the State Pharmacy Association to serve for a period of three years.

One (1) Registered Nurse elected by the State Nurses Association to serve for a period of three years.

One (1) Hospital Superintendent elected by the State Hospital Association to serve for a period of three years.

The above members of the Board of Governors shall elect for a period of three years two (2) additional members to represent the industries coöperating in Mutual Health Service and two (2) additional members to represent the recipients of health services.

3. Within such limitations as may hereinafter be provided, the general powers and duties of the Board of Governors shall be as follows:

a. The supervision of the administration of Mutual Health Service.
b. The appointment of administrative personnel.
c. The preparation of such rules and regulations as may from time to time become necessary to maintain or clarify the purposes of Mutual Health Service.
d. The preparation of fee schedules for the particular health services hereinafter described.
e. The administration and control of financial matters including the collection, expenditure and investment of funds.
f. The appointment of such committees as may be necessary to the proper functioning of Mutual Health Service.

4. Local direction in each county or district where Mutual Health Service is maintained shall be by the following committees and a District Mutual Health Committee:

a. Local Medical Committee.
b. Local Dental Committee.
c. Local Nursing Committee.
d. Local Hospital Committee.
e. Local Pharmacy Committee.

5. Each Local Committee shall be composed of five members elected or appointed for a period of three years by the body represented. In addition to such powers and duties as may hereinafter be provided the general functions of each Local Committee shall be as follows:

a. Local Medical Committee.
   1. The preparation of lists of general practitioners and of specialists for the provision of medical services.
   2. The control of the quality of medical service.
   3. The hearing of and action upon complaints involving physicians or physicians and patients.
   4. The reference, with recommendations, of those complaints involving a physician and a member of any other professional group to the District Mutual Health Committee.
   5. The transmission of recommendations dealing with financial matters to the District Mutual Health Committee.

(The powers and duties of other Local Committees representing professional groups would be comparable to the above.)

6. The District Mutual Health Committee shall be composed of the chairmen of the Local Committees, who shall elect one (1) additional member to represent the local industries coöperating in Mutual Health Service and one (1) member to represent the local recipients of health services.

The general powers and duties of the District Mutual Health Committee shall be as follows:

a. The hearing of and action upon complaints transmitted by Local Committees.
b. The transmission, with recommendations, of matters involving finances to the Board of Governors.

7. Any party involved in an action taken by a Local Committee shall have the right to appeal to the District Mutual Health Committee.

Any party involved in an action taken by the District Mutual Health Committee shall have the right to appeal to the Board of Governors.

8. The Board of Governors shall maintain a Local Executive Secretary and such other personnel as may be necessary in each District where Mutual Health Service is established. The Local Executive Secretary shall serve as executive secretary for each Local Committee and the District Mutual Health Committee.

9. The employee shall have the right to choose a general practitioner of medicine and a general practitioner of dentistry. There shall be no restrictions to such freedom of choice except as follows:

a. The general practitioner shall reside in the employee's city or within a reasonable distance from the employee's residence. "Reasonable distance" shall be determined by the Local Committee.
b. The general practitioner shall hold a license to practice his profession in Michigan.
c. The general practitioner shall have signified in writing his intention to provide service in accordance with the plan of Mutual Health Service.

10. The employee shall have the right to change his selection of a general practitioner of medicine or of dentistry in accordance with either of the following procedures:

a. By filing with the Local Executive Secretary a written notice of the intended change 14 days before any of the following dates: January 1, April 1, July 1, October 1, requesting a change on any of the above dates and naming the successor to his general practitioner.

b. By filing with the Local Executive Secretary a written request for an immediate change, stating the reason or reasons for the request and naming the successor to his general practitioner.

11. The general practitioner of medicine or of dentistry chosen by the employee shall have the right to accept or reject the application for his services. If he accepts he shall have the right to subsequently withdraw his acceptance in accordance with the following procedures:

a. By filing with the Local Executive Secretary a written notice of his withdrawal 14 days before any of the following dates: January 1, April 1, July 1, October 1.

b. By filing with the Local Executive Secretary a written request for immediate withdrawal stating his reason or reasons for such withdrawal.

12. The names of persons accepted for services by the general practitioners shall be known as the general practitioner's "Family List." The total numbers of persons on the Family List shall not exceed one thousand (1,000) for any one general practitioner; provided, however, that in those cases where a general practitioner has one or more salaried assistants licensed to practice in Michigan the number of persons on the Family List shall not exceed sixteen hundred (1,600). The District Mutual Medical Committee shall be empowered to increase the limit from 1,600 to 2,000 when, in its judgment, such extension or increase is necessary.

13. The general practitioner of medicine shall provide for the persons on his Family List such services as may be reasonably expected according to the standard of the community in which he practices his profession. These services shall include physical examinations, immunity tests, immunizations, prenatal and postnatal care and such services other than those designated as specialists' services; provided, however, that the general practitioner may include specialists' services in accordance with the regulations adopted by the Local Medical or the Local Dental Committees and approved by the Board of Governors.

14. Each person on the Family Lists shall be entitled to the following health service.

a. Home, office and hospital services of a general practitioner of medicine and such services of medical specialists, nurses, pharmacists, laboratories and hospitals as may, in the opinion of the general practitioner, be necessary; provided, however, that certain services shall be limited as follows:

1. Hospital services shall not be provided for mental diseases and tuberculosis.
2. Hospital services shall include a semi-private or ward bed, operating rooms, medicines, dressings, laboratory and other services that may be provided in the hospital for a period of 21 days during any one year. For any illness requiring hospitalization for more than 21 but less than 90 days, the Mutual Health Service shall pay 75 per cent of the per diem hospital charges.
3. The services of a special nurse shall be limited to a period of 30 days during any one year. The services of a visiting nurse shall be limited to 60 days during any one year.
4. Drugs shall be prescribed by a Doctor of Medicine to any member of the Family List at a cost of 25 cents for each prescription. The difference, if any, between this cost and the total cost of the prescription shall be paid by Mutual Health Service. Drugs shall be prescribed in accordance with such regulations as may be approved by the Local Medical and the Local Pharmacy Committees.
5. The care of any illness or accident provided for under the Workmen's Compensation Act shall be excluded from Mutual Health Service.

15. The costs of services shall be borne by the employee or jointly by the employee and his employer. Payments shall be made in advance either weekly, monthly, semi-annually or annually at a rate or rates established by Mutual Health Service.

16. For the period of the experiment the annual costs of services per person on the Family Lists shall be $27.88.

17. The annual sum of $27.88 per person on the Family List shall be expended as follows:

a. General Practitioner of Medicine	$ 5.00		
1. Report of Annual Physical Exam.	.50	31	%
2. Report of Immunization	.25		
b. Medical Specialists' Services	3.00		
c. Dental Services	5.00	18	%
d. Nursing Services	2.50	9	%
e. Hospital Services	5.00	18	%
f. Drugs, Medical, Surgical, Optical Appliances	2.00	7	%
g. Laboratory Services	1.00	3.5	%
Total	$24.25		
h. Administration, 10%	2.42	9	%
i. Surplus, 5%	1.21	4.5	%
Grand Total	$27.88	100	%

18. For each community or area in which Mutual Health Service operates, fee schedules shall be prepared for the following services shown in Paragraph 17:

1. Specialists' services in Medicine and Dentistry.
2. Nursing services.
3. Hospital services.
4. Drugs, Medical and Surgical Appliances.
5. Optical services.
6. Laboratory services.

The fee schedules shall be prepared by the Local Committees and transmitted through the District Committee to the Board of Governors for final action.

19. Any general practitioner of medicine or of dentistry whose qualification for the provision of any specialist service is approved by the Local Medical or the Local Dental Committee shall be entitled, in addition to the capitation fee, to 75 per cent of the specialist's fee as shown on the fee schedule.

The Local Committee shall have power to grant, reject or withdraw approval for specialist service or services by the general practitioner.

20. In the event that the cost for any service is proportionately higher than anticipated the total sum collected for that service shall be pro-rated among the individuals or institutions providing that service. In the event that the cost for any service is lower than anticipated, the excess shall be added to the Surplus or expended according to the judgment of the Board of Governors.

21. Surplus funds shall be maintained in separate accounts for each area or community and shall be expended or invested by the Board of Governors.

22. Two per cent (2%) of the sum allotted to the general practitioners of medicine and dentistry shall be deducted and placed in a "Postgraduate Training Fund." Expenditures from this fund shall be made by the Board of Governors with the advice of the State Medical and State Dental Societies.

The Committee feels that in the foregoing program there is presented a temperate policy for the guidance of the medical professions in the United States. It is a policy that preserves the essence of American practice in giving primary consideration to the potential consumers of health services. In this consideration those things that the profession has long cherished because they have been subjected to the retort of experience and have been found satisfactory are not only retained but strengthened. It is the Committee's opinion that the program presented is the studied and constructive answer of the profession to those encroachments upon private practice that have their origin in the unequal distribution of the services the profession commands.

If the House of Delegates approves the program, the Committee recommends that the action be given full publicity. Michigan is practically the only industrial state that remains comparatively free of numerous plans and projects for the provision of health services. This freedom can be attributed to no other factor than the action of the Michigan State Medical Society in undertaking constructive studies. It must remain free until the profession has had the opportunity to experiment. Nothing will insure this freedom as completely as publicity.

At the same time in many other states the profession finds itself in a difficult position. Lacking a constructive program it is easy prey for commercial or political influences. Publicity given to the Michigan program may, therefore, exert a far-reaching effect in providing the profession in other states with the methods and materials for combating destructive influences. Against these influences nothing is more effective than the sure weapon of constructive action.

If the program presented is acceptable to the House of Delegates, the Committee proposes the following steps in order to begin the experiment as early as possible:

1. The discussion of the proposals with employers and employees. Naturally the communities or areas selected for the experiment will depend upon the reception given to the plan by these groups.
2. The presentation of the final detailed plan to the House of Delegates.
3. The presentation of the plan to the professional groups in the area or areas selected for the experiment.
4. The formation of the local and state committees already mentioned.
5. The preparation of reports of the House of Delegates during the progress of the plan.
6. The preparation of a final report, with recommendations, at the completion of the experiment.

There are certain implications in the plan for Mutual Health Service that merit the attention of the House of Delegates. These deal with the relation of the plan to the care of indigents and to public health activities.

The Committee sees no reason why the plan should not be extended to include the care of indigents. It feels that with few exceptions the profession's method of handling this problem has been hesitant and generally ineffective as a result of the lack of a definite program. It is not necessary to defend the assumption that while a person may be designated as "unemployed and indigent" his health needs differ little from those of his more fortunate neighbor.

It is generally accepted that the responsibility for the health needs of indigent persons rests upon the community. Nor is the responsibility lessened because it has been shifted to the profession in certain communities. Therefore, the Committee recommends that the Local Committees engage in a joint effort to extend Mutual Health Service to indigents. The method of extension is for the community to enter into a contractual arrangement with Mutual Health Service. Such an arrangement will permit the unemployed and indigent person the same relationship to his general practitioner as he enjoys while employed.

The effect of Mutual Health Service upon public health activities is fairly obvious. In brief, it means an expansion of the services of preventive medicine and dentistry by private practitioners and a concentration of public health work upon educational activities. The Committee feels that such concentration will be heartily approved by the public health agencies concerned.

Thought should also be given by the Delegates to the probable development of a movement in the United States toward some form of compulsion with respect to the purchase or distribution of health services. There is little doubt that the movement has gained momentum and that the coming legislative sessions in many states will see bills presented for compulsory health insurance.

If the experiment with Mutual Health Service is successful the profession will be in an exceedingly strong position to direct public opinion and thereby control legislative action in the interests of public welfare. It is obvious that the advantage of controlling medical legislation will lie with the first constructive program presented. If, in addition to being first, the program has a record of successful demonstration, the advantage will become great enough to direct legislative action. Only in this manner may the profession hope to successfully combat commercialism and the profit-motive in the distribution of health services.

SUPPLEMENTAL REPORT OF THE COMMITTEE

As a supplement to its formal report your Committee presents certain additional items for consideration and action by the House of Delegates.

The first item deals with the matter of the upper limit of income and upon this question Dr. Sinai will present some data for the information of the delegates.

The second question deals with the association of official public health work with Mutual Health Service. It will be noted that public health is not represented on any of the Committees controlling Mutual Health Service. After some discussion of this aspect of Medical Care your Committee concluded that, if the general plan is approved, the House of Delegates should decide the following proposal:

That, in order to complete and unify all the health services in Michigan, the State Health Commissioner should be made a member of the Board of Governors and either the local or county health officer a member of the District Mutual Health Committee.

The third item deals with your Committee's suggestion for publicizing the program for Mutual Health Service.

In addition to its specific studies in Michigan your Committee has collected information on recent developments in medical care in other parts of the United States. Because of its knowledge of these developments, your Committee has suggested that if Mutual Health Service is approved by the House of Delegates, the plan be given as wide publicity as possible.

The purpose of this publicity is twofold: first, to refute the widespread charge that the profession is unwilling to apply the experimental method to any solution of its economic problems, and, second, to offer to the profession and the public a constructive program as a weapon against such harmful trends as are already developed or are in the process of development. Your Committee feels that Mutual Health Service, if adopted by the House of Delegates, is the unanswerable reply of the profession to the charge that it has failed to give studied consideration to the economic problems of medical care. It also feels that this "declaration of principles" is a standard around which both the public and the professions may rally in mutual protection and with mutual confidence.

A brief description of certain of the developments in the country will serve to substantiate the Committee's opinion that a constructive program is, at this time, a pressing need.

Many county societies have undertaken projects in health insurance with little study and with almost no central or state authority. The county societies cannot be criticized too greatly for this action. It has come in response to the profession's and the public's demand "to do something," and in most cases the state societies, being without any program, have been willing to permit the county societies to bear the brunt of any ill-advised action. This situation is particularly acute in the western states.

Your Committee feels that the major danger of this lack of state society direction lies in its tendency to develop commercial competition between groups of physicians and between other professional groups, as well. The results of such commercial competition have been evident for many years in the application of Workmen's Compensation in certain states. It was for this reason that your Committee stressed the comprehensive program with a centralized and professional financial control.

In this same connection, your Committee has noted the development of hospital insurance in over twenty states. There is nothing particularly wrong in the principle of hospital insurance. However, in the absence of a more comprehensive program your Committee feels that hospital insurance can expand in only one direction and that is to include other services. The demand for expansion has already arisen from the insured public. This means that, as additions to services occur, such additions become not a part of a generalized system but a part of hospital insurance and controlled by the agencies of hospital insurance.

Another significant development in this country is reported in the *New York Times* of March 16. The story dealt with the annual meeting of the advisory council of the Milbank Memorial Fund. At this meeting a plan for state controlled compulsory health insurance was presented and discussed. Those

present included Mayor LaGuardia and Harry L. Hopkins, Federal Relief Administrator. To quote from the *Times:*

"The plan," Dr. Miller said, "provides for local differences of ability to pay and for the availability of medical facilities by dividing the services into two classes. The first includes those which should be mandatory for the entire state. It would include home and office care by physicians, prescribed drugs and medicines, hospital care where the institutions are available, and maternity benefits for women who remain under continuous prenatal supervision.

"The second class includes those which are permissive and which may be established in local areas, with the approval of the proper authority, when the extension is desired by the local area, when the costs can be financed, and when the competent personnel and facilities are available. This would include specialist services, dentistry, the clinic and the laboratory, home nursing, etc."

* * *

"You aren't going to get health insurance," Mr. Hopkins said, "if you expect people to do it voluntarily. I am convinced that by one bold stroke we could carry the American people along not only for health insurance but also for unemployment insurance. I think it could be done in the next eighteen months."

Whatever may come as a result of the above advocacy, your Committee views Mutual Health Service as the organization with which state or other authorities must deal. The history of compulsory health insurance is a history of disastrous results to the professions because of a lack of unity. It is a history of an organized group dealing with disorganized professional units or with individuals.

A third development in the United States is the drive toward unemployment insurance. Among medical leaders there is a too general apathy toward this movement. If unemployment insurance becomes both widespread and inclusive, your Committee feels that thought must be given by the professions to two items: first, the setting aside of a percentage of unemployment insurance funds to provide medical care for the unemployed, and, second, the question of unemployment by reason of physical disability.

Out of the second item it is not improbable that methods may be developed for the care of physically unemployable people so that they may again become employable. The history of Workmens' Compensation shows that the profession gave little or no thought to its medical aspects. In this connection the following quotation from the report of the Committee on Legislation and Public Policies in *The Journal of the Michigan State Medical Society* for March, 1912, will be of interest:

"True we have had two sessions called by the Governor to consider special and specific subjects; yet at these sessions nothing of a medical or public health nature was considered. Nothing at these sessions could be legally or constitutionally considered other than those recommended from time to time by special message from the Governor to the Legislature. Therefore, if any amendments to our medical laws are to be made they must be made during the next regular session of the Legislature which will convene January, 1913."

It was at one of the special sessions where "nothing of a medical or public health nature was considered" that Michigan adopted the law for Workmen's Compensation. Your Committee presents this item for whatever may be its value in focusing attention upon unemployment insurance.

As reported to the House of Delegates at its last meeting, the California legislature has appointed a commission to study health insurance and report in January, 1935. No further information concerning the activities of the commission is available. The whole subject, however, is on the program of the Western Hospital Association meeting in Sacramento, California, this week, and also at a meeting to be held before the Commonwealth Club in San Francisco on Friday.

Recently the Committee on Medical Economics of the Colorado State Medical Society published a report advocating the application of the principle of health insurance. To quote from this report:

"The point at issue is not the insurance principle but how to use it.

"We are not unmindful of the opposition of the A. M. A. to the general plan of insurance medicine, especially as operated by European governments or by corporations for profit; nor unmindful of the deteriorations accompanying contract practice regardless of how disguised. The plan referred to is not in the opposed classes because it does not restrict opportunity to practice, nor free choice of physician, is on a non-profit basis and under the control and management of the profession. This places the responsibility where it belongs. All of us have looked to the A. M. A. for help. Thus far we have been disappointed."

An interesting booklet has been received recently by your Committee. It contains the platform and principles of the Medical League for Socialized Medicine, an organization with headquarters in Brooklyn and with a membership of over 500 physicians. This organization is unequivocally committed to the principle of salaried physicians providing service under the state.

These are a few of the evidences of the turmoil in medical economics. It is for its quieting and beneficial influence that your Committee has recommended that the plan of Mutual Health Service be widely publicized.

RESOLUTION OF APPROVAL

Whereas, the House of Delegates of the Michigan State Medical Society, at its meeting on July 12, 1933, directed the Committee on Medical Economics to prepare and present for consideration of

the House a plan or plans for health insurance; and

Whereas, the House of Delegates adopted the policies for the guidance of its Committee on Medical Economics:

and

Whereas, the plan for Mutual Health Service presented by the Committee on Medical Economics is in accord with the above policies; therefore, be it

Resolved, that the House of Delegates of the Michigan State Medical Society approves the general principles of the plan for Mutual Health Service, and directs the Committee on Medical Economics to undertake the following efforts:

a. The discussion of the plan with employers and employes.
b. The determination of the legal status of the Mutual Health Service, and the necessary legal action for the organization of Mutual Health Service.
c. Preparation of the final detail plan of the Mutual Health Service and its presentation to the House of Delegates for final action;

and be it further

Resolved, that the plan for Mutual Health Service shall not be inaugurated in any county without the approval of the county and state medical society.

AN HONOR TO DR. GEORGE GILBERT SMITH

At the meeting of the American Urological Association, held at Atlantic City, May 25, Dr. George Gilbert Smith of Boston was elected president. Dr. Smith is a member of the staff of the Massachusetts General Hospital, Department of Urology.

CORRESPONDENCE

The Massachusetts Medical Society

POSTGRADUATE INSTRUCTION

May 26, 1934.

Editor, *New England Journal of Medicine*,

On May 15, 1934, the Massachusetts Medical Society's first year of organized postgraduate extension courses came to a close. One thousand and two Fellows were enrolled which may be considered a successful beginning. The Executive Committee has prepared a report of the year's work which will be submitted to the Council on June 5.

Some comments by those who took the course may be of interest.

1) "Looking forward to next year's lectures."
2) "I want practical everyday talk, not too many statistics."
3) "Course worth while. All members of Massachusetts Medical Society should be compelled to take this course."
4) "Suggest an outline of year's lectures be mailed each doctor in advance."
5) "I believe the course was too elementary."
6) "Most excellent undertaking and would greatly appreciate more."
7) "Well worth while. Valuable and stimulating as such things always are when given by good men."
8) "The choice of speakers was happy—men who were practical rather than didactic."
9) "Make them more useful and practical for the family physician."
10) "I favor more questions; some of the points that helped me most were unearthed by questions."
11) "In my opinion postgraduate teaching does not mean reading papers. Each instructor should come with his subject prepared so that he can stand up and give it without notes or with notes. I feel that the instructor should not attempt to cover too much ground."
12) "I think the courses were too general; would prefer more details of diagnosis and treatment."
13) From a district chairman: "First, in my opinion this was one of the finest courses ever afforded to the physicians in our County, and I am convinced from many remarks made to me from the majority of the men, and from men whose opinions I value most, that the course was appreciated and will be looked forward to in the years to come. I am sure next year we will have a much larger turnout."
14) From another district chairman: "The talks have been very excellent in every way and have been much enjoyed by all of the men, and they feel that it is one of the best things that the Massachusetts Medical Society in association with the District Societies has ever put over. It has done much to bring the men together, and to promote harmony and good fellowship, and this is a very important thing in any community undertaking. The local men have tried to express their appreciation in various ways for the efforts that have been made by the different speakers in making such long trips to carry out this program."

The following letter from a Rhode Island doctor who took the course at Fall River is of interest:

"I had the opportunity to attend the lectures sponsored by the Massachusetts Medical Society, on specialized medical subjects, given at Fall River. I take this occasion for congratulating the Society for such a step forward.

"All the lecturers were very interesting and they all presented their respective subjects in such a way that the important facts were emphasized. I need not say any more because these physicians are already known.

"I sincerely hope that this will become a tradition, and that next year we shall again enjoy the same privilege of being taught the best things by the best men."

The program for next year will be published before the autumn. Anyone interested in special subjects should consult his district chairman on postgraduate instruction. The final report will be posted in the exhibit space at the Worcester Convention.

Sincerely yours,

LEROY E. PARKINS, M.D., *Secretary,*
Executive Committee
on Postgraduate Instruction.

THE NECESSITY FOR THE JUDICIOUS USE OF ARTIFICIAL PNEUMOTHORAX

May 24, 1934.

Editor, *New England Journal of Medicine.*

In the *Journal* of May 17, 1934, Drs. Walter C. Bailey and Bayard T. Crane of the Central New England Sanatorium called attention to the physicians of New England that they will accept cases of early tuberculosis for *deflation* treatment without cost of any kind to the patient. They propose to keep these patients at the sanatorium for ten days or two weeks and then return them to the family physician for refills and further care. By deflation I suppose they mean artificial pneumothorax treatment and in this way suggest that pneumothorax treatment should become the universal practice of every family physician. They do not mention any period of observation prior to instituting pneumothorax and infer that every patient should be ready to go back to his or her home after a brief stay of ten days or two weeks.

Those of us who have had some experience in the treatment of pulmonary tuberculosis do not as yet feel that collapse therapy should be used in every instance. Artificial pneumothorax is only one form of collapse therapy. A goodly number of patients if found in the early stages will do well on sanatorium régime alone. They make no mention of selecting patients, but will abide by the family physician's order for pneumothorax treatment. As yet I feel that collapse treatment is only an adjunct, to be sure a very important one, to the general treatment, and should be distinctly in the hands of those who are experienced in the general treatment of tuberculosis.

While the technical part of this treatment is a simple one, untold damage may be done by its injudicious use. The problem of the effectiveness of this treatment is a very serious one. The question of dosage is of extreme importance, and the question also of intervals is a very serious one to decide. Then there are always complications to treat, such as effusions.

The mere compression of the lung does not necessarily mean that the affected lesion has been well taken care of, adhesions frequently preventing a good collapse. Other forms of collapse therapy, if pneumothorax treatment fails, must be considered. A shifting mediastinum may also become a very irksome problem, the whole lung may simply be pushed over rather than compressed. Frequent fluoroscopic examinations before and after refills are of the greatest importance. The technical procedure itself is not without its real dangers. Lungs have been punctured and air emboli may occur. The length of treatment is a very delicate and a very important problem. Space will not allow enumeration of the many factors that will make for the success or failure in the treatment of pulmonary tuberculosis.

It has been a hard battle to bring the profession to the realization of the importance of collapse therapy in pulmonary tuberculosis. Let us not go too far now and destroy a method of treatment which has been a real blessing to the tuberculous patient by its injudicious, untrained and inexperienced use.

Sincerely yours,

MOSES J. STONE, M.D.

FELLOWSHIPS AT THE HARVARD MEDICAL SCHOOL

To the Editor:

The Commonwealth Fund of New York has added a course in office surgery to the list of subjects which are available for fellowships at the Harvard Medical School, Courses for Graduates. This course is designed for physicians who are engaged in general practice. The subjects studied will be surgical problems met in office practice. The course is for one month.

The other courses for which fellowships are available are medicine, obstetrics and pediatrics. Fellowships are for one to four months. The stipend is two hundred and fifty dollars per month, plus tuition and traveling expenses from place of residence to Boston and return.

An applicant for a fellowship must be a graduate of a grade A medical school; a member of the Massachusetts Medical Society, in good standing; must have been in practice at least five years; should be between thirty and fifty years of age; and resident of a town of under ten thousand population.

Application blanks may be obtained from the Commonwealth Fund, 41 East 57th Street, New York City, or from the Courses for Graduates, Harvard Medical School, 25 Shattuck Street, Boston.

Sincerely yours,

LEROY E. PARKINS, M.D.,
Secretary to Courses for Graduates under the Commonwealth Fund.

RECENT DEATHS

SMITH—Joseph Andrew Smith, M.D., of 584 Cambridge Street, Worcester, Massachusetts, with an office at 476 Main Street, died in that city, April 16, 1934. He was born in Worcester in 1875, and was educated at the Worcester public schools, Holy Cross College, and the Philadelphia College of Dental Surgery. His M.D. degree was conferred by the College of Physicians and Surgeons of Boston in 1914. His hospital training included two and a half years in the out-patient department of the Boston City Hospital. He joined the Massachusetts Medical Society in 1920, and specialized in oral surgery.

He served as police surgeon for several years, and had an extended military service, first as an enlisted private in the Spanish-American War, and was assigned to Fort McPherson, Georgia, Camp Hobson, Georgia, and Plattsburg, New York. On returning to Worcester he was appointed as paymaster of the First Battalion, M. V. M., with the rank of lieutenant and later promoted to that of captain, and retired from service with the rank of major. He was very much interested in the Boy Scouts and Red Cross activities, and was prominent in politics. He served on the Worcester school board. He was a member of Alhambra Council, K. of C., the Eagles, Division 25, A. O. H., E. R. Shumway Camp, United States War Veterans, the Economic Club, McCafferty Court, M. C. O. F., the Electric Medical Society, the Worcester District Dental Society, and was a founder of the St. Vincent de Paul Society of Sacred Heart Church.

He is survived by his widow Mrs. Mary J. (Dunn) Smith, and three children, Mary, Joseph, and Lewis A.; two brothers, Rev. Thomas P. Smith of Blackstone, and Rev. William F. Smith of Oakdale; and a sister, Miss Julia Smith of Worcester.

HILL—George Hilliard Hill, M.D., of 15 High Street, Worcester, Massachusetts, died in that city, May 18, 1934, after an illness of several months, terminating in pneumonia. He was born in Worcester in 1868, the son of J. Henry Hill and Sarah Bruce (Jenkins) Hill. He was educated in King's private school, Leicester Academy and graduated from the Harvard Medical School in 1894. He first practiced in Millis and moved to Worcester in 1897. He served on the staffs of the Memorial and the Worcester City Hospitals, but resigned from hospital service several years ago, devoting much of his time to the duties of surgeon to the Worcester Fire Department, reporting at all fires, even at times when his health had become seriously impaired.

He joined the Massachusetts Medical Society in 1896, and the American Medical Association the same year. He was a Fellow of the American College of Surgeons, and a member of the Harvard Club of Worcester, Devens Post, A. L., the Leicester Golf Club, and Cohasse Golf Club of Southbridge.

He is survived by his widow, Grace R. E. (Elliott) Hill, and three nephews, Southworth Lancaster, Bruce Lancaster, and Eveleth D. Hill, all of Worcester.

NEWTON—Edward Roswell Newton, M.D., of Brookline, Massachusetts, with an office at 419 Boylston Street, Boston, died at his home, May 19, 1934, after a long illness. He was born in Hartford, Connecticut, in 1874, and graduated in medicine from the Harvard Medical School in 1898. After postgraduate studies in Berlin, Vienna and Paris in diseases of the nose, throat, and ear, he practiced in Boston.

During the World War he served in the United States Navy and practiced his specialty.

He joined the Massachusetts Medical Society in 1914. He was a Fellow of the American Medical Association and a member of the American Otological Society. He is survived by his widow, Mrs. Mabelle M. (Field) Newton, three brothers, and two sisters.

DENIG—Blanche A. Denig, M.D., formerly of Boston, and lately living at Oakland, California, died recently.

She was born in Columbus, Ohio. While in Boston she had offices at 541 Boylston Street, and was a member of the staff of the New England Hospital for Women and Children.

OSTROM—Hjalmar Ostrom, M.D., of Quincy, Massachusetts, with an office at 419 Boylston Street, Boston, died at the New England Baptist Hospital, May 24, 1934.

Dr. Ostrom was born in Sweden in 1882, and came to the United States when he was seventeen years of age. He studied for three years in the Morgan Park Theological School, and graduated in medicine from the Creighton University School of Medicine in 1911. Soon after graduation he served as a missionary to the Belgian Congo for seventeen years representing the American Baptist Foreign Missionary Society. While there he was decorated by the late King Albert for meritorious service both as a missionary and for his medical work in combating disease in that territory. During a leave of absence he studied tropical diseases at Harvard College; London, and Brussels. He retired from the foreign field and engaged in practice in Quincy in 1929. He joined the Massachusetts Medical Society in 1930.

Dr. Ostrom is survived by his widow Mrs. Agnes (Brodd) Ostrom, and two daughters, Miss Mary Ann and Miss Helen Ostrom.

SIMMONS—Fred Albert Simmons, M.D., 101 Bay State Road, Boston, Massachusetts, died at his home, May 27, 1934, after an illness of two days.

Dr. Simmons was born in North Adams in 1877, and was educated in the public schools of Adams, and at Brown University, graduating with the de-

gree of Ph.B., and received his M.D. degree from Columbia University College of Physicians and Surgeons in 1903. He practiced in Brockton, Massachusetts, for several years, later settling in Boston where he specialized in otology, laryngology and rhinology, and was formerly an instructor in otology at the Harvard Medical School, and associate surgeon at the Massachusetts Eye and Ear Infirmary, and associate surgeon at the Massachusetts General Hospital. He served as corporal in the Spanish-American War, and was a member of the medical advisory board during the World War.

He joined the Massachusetts Medical Society in 1905, and was a Fellow of the American Medical Association, a Fellow of the American Board of Otolaryngology, and a member of the New England Otological, Laryngological and Rhinological Society.

He belonged to the Commercial Club, the University Club, the Thorney Lean Golf Club of Brockton, and the Harvard Club. He was prominent in the Masonic fraternity and the Knights of Pythias.

Dr. Simmons is survived by his widow, Mrs. Geneva (Cobb) Simmons; one son, Dr. Fred A. Simmons, Jr.; two brothers, Louis A. Simmons, of North Adams, and Arthur C. Simmons of Adams, and a sister, Miss Edith M. Simmons, of Adams.

NOTICES

The Massachusetts Medical Society

ANNUAL MEETING

PROGRAM FOR THE ENTERTAINMENT OF THE VISITING WOMEN

Monday, June 4.

9 A.M. to 12 M. The local women's committee will be at the Hotel Bancroft to welcome and assist the visitors.

12 M. A bus will take visitors to the following:

The Library of the American Antiquarian Society.

Tatnuck Country Club for luncheon at one o'clock.

The J. W. Higgins Armory Museum.

The Worcester Art Museum.

The Worcester Club for tea about 4:30.

8 P.M. The Shattuck Lecture. Hotel Bancroft, Worcester.

Tuesday, June 5.

9 A.M. to 12 M. The local women's committee will be in the hotel lobby to welcome and assist the visitors.

12 M. A bus will take visitors to the following:

The Worcester Memorial Auditorium.

The Worcester Country Club for luncheon at one-thirty o'clock.

The gardens of Mrs. Homer Gage in Shrewsbury at about three o'clock.

7 P.M. The Annual Dinner.

The galleries of the ballroom have been reserved for ladies who wish to hear the distinguished after-dinner speakers.

REMOVAL

M. A. WEINGER, M.D., announces the removal of his office from 371 Commonwealth Avenue to 395 Commonwealth Avenue, Boston.

REPORTS AND NOTICES OF MEETINGS

OFFICERS OF THE NEW ENGLAND PHYSICAL THERAPY SOCIETY

At the annual meeting of the New England Physical Therapy Society held May 16, 1934, the following officers were elected: Dr. Charles W. McClure of Boston, president; Dr. George B. Carr of Lynn, and Dr. George B. Rice of Boston, vice-presidents; Dr. Arthur H. Ring of Arlington, secretary; Dr. Franklin P. Lowry of Newton, treasurer.

The councilors are, for three years, Dr. William G. Curtis, Wollaston, and Dr. DeWitt G. Wilcox, Newton; for two years, Dr. John L. O'Toole, Haverhill, and Dr. Solon Abbott, Franklin; for one year, Dr. William D. McFee, Boston, and Dr. John F. Valentine, Danvers.

Dr. William L. Clark, Philadelphia, was elected to Honorary Membership in the Society.

MASSACHUSETTS SOCIETY OF EXAMINING PHYSICIANS

At the meeting of the Massachusetts Society of Examining Physicians, May 23, 1934, Dr. J. E. Goldthwait delivered an address under the title of "Chronic Invalidism Due to Joint Pathology." The discussion was opened by Dr. Howard Thompson who spoke on the medical aspects of the subject. He was followed by Dr. Carl Watson who presented the osteopathic point of view.

The officers elected for the ensuing year are:

Dr. Frederick W. O'Brien of Boston, president; Dr. George H. R. Gosman of Waban, Dr. James R. Knowles, and Dr. Bernard A. Godvin of Boston, vice-presidents; Dr. William Pearce Coues of Brookline, secretary, and Dr. Robert C. Gwin of Brookline, treasurer. The following were elected councilors: Dr. Francis R. Mahony of Lowell, Dr. John W. McNamara of Brockton, and Drs. Arial W. George, William F. Cotting, Samuel Tartakoff, Michael E. McGarty, George W. Morse, all of Boston.

THE ANNUAL MEETING OF THE NATIONAL TUBERCULOSIS ASSOCIATION

Seven hundred and sixty-six persons from all parts of the country registered at the Thirtieth Annual Meeting of the National Tuberculosis Association

held at Hotel Netherland-Plaza in Cincinnati, Ohio, on May 14, 15, 16, and 17.

The American Sanatorium Association and the National Conference of Tuberculosis Secretaries also met at the same place on Monday, May 14.

Those in attendance from Massachusetts included Dr. Henry D. Chadwick, State Commissioner of Public Health; Dr. Sumner H. Remick, Superintendent, Middlesex County Sanatorium; Dr. Edson W. Glidden, Superintendent, Worcester County Sanatorium; Dr. Olin S. Pettingill, Superintendent, Essex County Sanatorium; Miss Ida Cannon, Chief, Social Service Department, Massachusetts General Hospital; Dr. John B. Hawes, 2nd, President, Boston Tuberculosis Association; Miss Jean V. Latimer, Educational Secretary, and Frank Kiernan, Executive Secretary, Massachusetts Tuberculosis League.

Dr. John B. Hawes, 2nd, gave a paper in the Clinical Section on "Dust and Its Relation to Tuberculosis." Dr. Henry D. Chadwick was called upon to comment on papers in the Clinical, Pathological and Sociological Sections. Miss Jean V. Latimer spoke at the Health Education Meeting of Tuberculosis Secretaries. Mr. Frank Kiernan, who was Chairman of the Sociological Section, presided at the sessions on Tuesday and Thursday and also spoke on the program of the National Conference of Tuberculosis Secretaries.

Dr. H. Kennon Dunham of Cincinnati, Ohio, was elected President of the National Tuberculosis Association for 1934-35; Dr. Edward S. McSweeney of New York was elected President of the American Sanatorium Association; and Mr. Ernest D. Easton of Newark, New Jersey, was elected President of the National Conference of Tuberculosis Secretaries. All three organizations will come together again in June, 1935, for their Annual Meetings at Saranac Lake, New York.

NEW ENGLAND PHYSICAL THERAPY SOCIETY

The June meeting of the New England Physical Therapy Society will be held at the Hotel Bancroft, Worcester, in conjunction with the program of the Section of Radiology and Physical Therapy of the Massachusetts Medical Society at 2 P.M. on Monday, June 4.

This will be the final gathering of the year.

All members of our Society should make an effort to be present.

ARTHUR H. RING, M.D., *Secretary.*

BOSTON SOCIETY OF ANESTHETISTS

The next meeting of the Boston Society of Anesthetists will be held in the Hotel Kenmore on Tuesday, June 12.

The program:

6:30—Dinner, main dining room.

7:45—Business meeting, Colonial Room.

8:00—Scientific program, Colonial Room. Dr. Will Sentman Taylor, professor of psychology at Smith College, will talk on "Hypnotism: Its Relation to General Medicine and to Anesthesia," and will induce hypnosis in a subject.

Physicians and medical students are invited.

PHILIP D. WOODBRIDGE, M.D., *President*

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, *June 12*, 1934, at 12 noon. This is the *second Tuesday* of June. The meeting is being held at this time because the third week in June is Commencement Week, and it would be impossible for many to attend. The speaker, June 12, will be Dr. Tracy B. Mallory, Instructor in Pathology at Harvard Medical School; Chief of the Laboratory of Pathology and Bacteriology at the Massachusetts General Hospital. His subject will be "The Pathology of Bronchial Asthma." All physicians are cordially invited to attend both lecture and luncheon.

FAULKNER HOSPITAL CLINICAL MEETING

The final clinical meeting of the season will be held on Thursday, June 7, at 5:00 P.M., at the hospital. In addition to the usual clinical pathological conference Dr. Edward L. Young, Jr., who was scheduled to speak at the April meeting, but was unable to do so, will discuss "Some of the Difficulties of Gallbladder Diagnosis." All physicians who are interested are cordially invited.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

May 31—New England Medical Society will meet in the Evans Auditorium of the Massachusetts Memorial Hospitals, 80 East Concord Street, Boston, at 8:15 P.M.

May 31 - June 2—American Association for Thoracic Surgery. A program of papers on thoracic surgery and allied subjects will be given on Thursday and Friday in Building C Amphitheatre at the Harvard Medical School. On Saturday morning programs will be provided at the Massachusetts General Hospital, the Peter Bent Brigham Hospital, and the New England Deaconess Hospital on thoracic disease.

June 4—New England Physical Therapy Society. See notice elsewhere on this page.

June 4—Worcester City Hospital Clinics. See page 1137, issue of May 24.

June 4—St. Vincent Hospital (Worcester) Clinics. See page 1137, issue of May 24.

June 5—Alumni Luncheon, Massachusetts Alumni of the University of Maryland Medical School, Baltimore Medical College, College of Physicians and Surgeons, Baltimore, to be held at the Hotel Bancroft, Worcester, at 12:30 P.M. Reservations should be made through Dr. W. B. Davidson (1917), 36 Pleasant Street, Worcester.

June 7—Faulkner Hospital Clinical Meeting. See notice above.

June 7, 8, 9—Annual Meeting of the American Association for the Study of Goiter will be held in Cleveland, Ohio. For details of the program apply to Dr. J. R. Yung, Terre Haute, Ind.

June 11—American Medical Golfing Association will meet at the Mayfield Country Club in Cleveland. For details see page 1090, issue of May 17.

June 11-12—The American Proctologic Society will meet at the Hotel Cleveland, Cleveland. For information address the Secretary, Frank G. Runyeon, M.D., 1361 Perkiomen Avenue, Reading, Pa.

June 12—South End Medical Club. See notice above.

June 12—Boston Society of Anesthetists. See page 1196.

June 12—American Heart Association will meet at 9:30 A.M. at the Cleveland Hotel, Cleveland, Ohio.

July 24-31—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schnig. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

August 18 - September 30—Medical Study Trip to Hungary. See page 975, issue of May 10.

September 3-6—American Public Health Association, at Pasadena, California. Dr. J. D. Dunshee, Chairman, Local Committee on Arrangements.

September 4, 5, 6—International Union Against Tuberculosis. For information address the National Tuberculosis Association, 450 Seventh Avenue, New York City.

BOOKS RECEIVED FOR REVIEW

You Must Relax. By Edmund Jacobson. 201 pp. New York: McGraw-Hill Book Company. $1.50.

Medicine. A Voyage of Discovery. By Josef Löbel. 334 pp. New York: Farrar & Rinehart, Inc. $3.00.

Japanese Medicine. By Y. Fujikawa. Translated from the German by John Ruhräh. 114 pp. New York: Paul B. Hoeber, Inc. $1.50.

Survey of Public Health Nursing: Administration and Practice. By the National Organization for Public Health Nursing. 262 pp. New York: The Commonwealth Fund. $2.00.

The Hospital Manual of Operation. By Warren P. Morrill. 305 pp. New York: Lakeside Publishing Company. $3.00.

Brucella Infections in Animals and Man. Methods of laboratory diagnosis. By I. Forest Huddleson. 108 pp. New York: The Commonwealth Fund.

Surgical Clinics of North America. April, 1934. Volume 14—Number 2. New York Number. 520 pp. Philadelphia and London: W. B. Saunders Company.

Acute Intestinal Obstruction. By Monroe A. McIver. 430 pp. New York: Paul B. Hoeber, Inc. $7.50.

The Road to Adolescence. By Joseph Garland. 293 pp. Cambridge: Harvard University Press. $2.50.

Diseases of the Skin. For the use of students and practitioners. By Oliver S. Ormsby. Fourth Edition, thoroughly revised. 1288 pp. Philadelphia: Lea & Febiger. $11.50.

Modern Drug Encyclopedia and Therapeutic Guide. By Jacob Gutman. 1393 pp. New York: Paul B. Hoeber, Inc. $7.50.

One Hundred and Twentieth Annual Report of the Trustees of the Massachusetts General Hospital, including the General Hospital in Boston and the McLean Hospital in Waverley. 1933.

BOOK REVIEWS

External Diseases of the Eye. By Donald T. Atkinson. 704 pp. Philadelphia: Lea & Febiger. $7.00.

Atkinson's "External Diseases of the Eye," a book of 674 pages and ample index, abundantly illustrated with 479 engravings, will appeal to the general practitioner who must refresh his memory about diseases which affect the anterior segment of the eyeball, and acquaint himself with the methods of treatment employed.

Well-taught and recent medical graduates will be disappointed to find so little about the bacteriology and histo-pathology of ocular lesions, about the valuable diagnostic information obtained from the anterior segment with the use of the corneal microscope and slit-lamp, and about the supreme value of careful perimetry in following the progress of glaucoma of all sorts. The excellent arrangement of the subject matter, and the abundance of illustrations will make the book a useful one for orientation of the general practitioner.

Hygiene of the Mind. By Baron Ernst von Feuchtersleben. Translated from the German by F. C. Sumner. 150 pp. New York: The Macmillan Company. $1.25.

This translation of a little book of essays by a German physician who was evidently intensely human in his attitude, though written a hundred years ago, is of interest today to the doctor who views his patients as thinking living organisms, rather than as intricate machines. The Baron with prophetic insight saw beneath the surface of diseased segments into the minds which, in the last analysis, control those organs. His refreshingly individual philosophy is not far out of line with the thought of the most modern psychologists.

Surgical Clinics of North America. February, 1934. Volume 14, Number 1. Philadelphia Number. Clinic Year (February 1934 to December 1934) 226 pp. Philadelphia: W. B. Saunders Company. Paper, $12.00; Cloth, $16.00.

The principal feature of this number is a group of clinics on foreign bodies in the gastro-intestinal tract by Shallow, Clerf, and Manges. Foreign bodies in the esophagus or stomach can be removed endoscopically preferably with the aid of biplane fluoroscopy. Those which remain persistently lodged in the duodenum usually require operative removal, while those that pass the duodenojejunal junction are usually passed naturally.

Eliason and McLaughlin present an analysis of thirty-two cases of postoperative pulmonary atelectasis with but three deaths. Although this report is from a bronchoscopic center, only two cases were subjected to bronchoscopy.

The meticulous care necessary in the treatment of chronic laryngeal stenosis is outlined by Jackson. Some cases can be satisfactorily dilated while others require careful plastic surgery.

The other clinics by leading surgeons of Philadelphia are well chosen to cover subjects that are sufficiently common to be of real practical value to the average surgeon.

hoped that the rather dismal picture of medical education is not true of all medical schools.

Nevertheless stimulating ideas often come from fields lying outside of one's formal major interest and there is need that teachers of medicine should become conscious of developments in the field of pedagogy. For the inexperienced teacher this is especially helpful, and there are in Dr. Reid's book many useful hints, suggestions and directions. These are overlaid perhaps a little too much with the patter of the educationalists, and form a compilation of maxims rather than an integration of ideas about education. The book notes types of experiments among which the inexperienced teacher may find something suited to his immediate need. It does not supply a philosophy of education in the light of which procedures may be judged.

But the author claims in his preface that his book is only "suggestions." Accepting this statement rather than the more ambitious title as a criterion by which to judge accomplishment, the book may serve as a finger-post to direct teachers of medicine to investigations of educational procedure so that they may find for themselves something by which they may become better teachers. The secret of excellent teaching is to have an excellent teacher. This fundamental consideration in education the book does not discuss.

Social Psychology. By Abraham Myerson. 640 pp. New York: Prentice Hall, Inc. $3.50.

This comprehensive volume includes all the various aspects of social psychology both from the descriptive and dynamic viewpoints. The fact that it is written by a physician and a trained psychiatrist of long experience and keen insight makes the volume of more practical value than similar treatises by non-medical psychologists who tend to delimit and isolate the various determinants of group psychology. Dr. Myerson has been able to integrate and evaluate the neurological basis of human behavior, because of his opportunities to observe and study the effects of the visceral-organic structure upon the total personality.

The approach to the various aspects of social psychology is based upon two main theses, namely, that the visceral-organic structure of man is basic for an understanding of psychology and secondly, that apart from his group, man is a mere potentiality, it being necessary for him to develop in an environment which may foster, modify or even destroy his capacities. The student will find in this volume all the general material for the orientation of his studies with the more specialized fields of the social sciences, particularly in the various discussions and detailed bibliographical references.

The book is divided into four parts: general social psychology, visceral social psychology, social psychology of the family and the social aspects of crime. It is emphasized that even individual psychology is social psychology, because the group is an integrated whole and not a broken mosaic of isolated units.

Within the limits of a review, it is possible to indicate only briefly the rich material so well assembled and analyzed in this volume. It is refreshing to see the reflex as a unit of behavior, criticized, as such objective-experimental methods are inadequate to explain the complexities of human or even animal mental processes. One of the most interesting sections is that devoted to crime, particularly the description of that criminal type of minor delinquents which the author terms "unorganized extroverts," that is, individuals whose activities do not follow an organized or integrated plan. Sexuality and the digestive processes, as the two most important emotional drives, are visceral-social affairs and are given an extended discussion because both enter psychologically and physiologically into human conduct, influencing mood, emotional satisfaction and even symbolization and morality. In fact, in the reproductive processes, one is not dealing with a sexual instinct but with a psycho-sexual one; even character traits show this linking of mental and physical from gastro-intestinal activities, as shown by the reconstruction of the oral and anal character as formulated by psychoanalysis. The social structure continually modifies, inhibits and excites what is termed the "curiosity mechanism". It does this by means of diversified receptors which organize all individual experience. This learning-teaching mechanism is synonymous not only with intelligence, but extends to every visceral function, developing social attitudes and the dominance of the reality principle.

The volume can be highly recommended, not only for its lucid analyses and keen insight, but also because the various approaches as developed by the author are so stimulating and provocative, that the reader will feel strongly inclined to pursue the various avenues of the subject.

Treatment in General Practice. By Harry Beckman. Second Edition. 889 pp. Philadelphia: W. B. Saunders Company. Cloth, $10.00 net.

The reviewer of this volume must confess to a previous bias against textbooks devoted especially to such subjects as prognosis, therapeutics, treatment, etc. Perusal of Professor Beckman's unusual work does much to dispel this bias. In well-written, often sparkling English the results are presented of a pharmacologist's investigations into the literature relating to the modern techniques of therapy. It is quite true, as the author states in his preface, that the subject of therapy is inadequately taught and that general rather than specific principles are emphasized. So much the more important is it therefore for the practitioner to possess a volume of this sort, in which the last word on the treatment of a given disease is presented in detail. To be sure, this material may be obtained from the original articles, but these are scattered in so many journals that

their accessibility frequently becomes difficult or impossible to the average man. Beckman reads all the articles, classifies them, examines them critically, and finally presents them to the practicing physician ready for immediate service in the case at hand. A possible criticism is that multiple methods of treatment are frequently presented without an outline of what the author considers is the best program of therapy. This may be valuable to the initiated but must be rather difficult to the novice.

The book is startlingly up-to-date: witness the inclusion of a section regarding the possible dangers of amidopyrine and related drugs in agranulocytosis, a recital of the doses of the various preparations of liver for parenteral use, etc. The author's digestion of so many articles on so many diverse subjects does not appear to have influenced his critical faculties for his observations remain throughout quite sane. The most valuable feature of the book is its exact detailed description of the methodology of treatment, often given in the direct words of the authorities quoted. A section on "vehicles and incompatibilities," an alphabetically arranged bibliography of about 2500 references and an excellent index complete the volume.

The Technique of Local Anesthesia. By Arthur E. Hertzler. Fifth Edition. 292 pp. St. Louis: C. V. Mosby Company. $7.50.

Hertzler aims to present briefly and simply his own technique of local infiltration anesthesia and to lay very little emphasis on the methods of regional anesthesia. He appears to draw from profitable and very extensive personal experience. By mingling descriptions of surgical methods with local anesthesia technique, he points out how operations may be adapted to the demands peculiar to local anesthesia. He has a sane realization of the limitations of local blocking, and with each region of the body discusses without prejudice the relative merits of that method and of general anesthesia. This is one of the most distinctive and valuable characteristics of the book.

Although he stresses the importance of accuracy of technique and the use of small amounts of a solution, yet unfortunately in the detailed directions he too frequently omits mention of the amounts to be used. He discards nupercain from consideration, quite properly, but defends the use of quinine and urea hydrochloride. His unreserved recommendation of diothane, a new drug of questionable merit, appears ill-advised. There is no mention of the prophylactic use of the barbiturates, nor of the treatment of procaine toxicity.

The illustrations are of good quality and number, but unfortunately some of them are not labeled. Detailed instructions are clear and adequate on many subjects, notably inguinal and umbilical hernias, the bladder, the male and female genitalia, the rectum, the branches of the fifth cranial nerve, and the paravertebral injection of the thoracic nerves. The general discussion concerning abdominal anesthesia is excellent. It includes the indications for local anesthesia, the neurological anatomy involved, and the sensitiveness of the viscera to various types of stimulation under varying degrees of inflammation. He agrees with others in placing little dependence on splanchnic anesthesia. It is disappointing to find that he has nothing to offer in the way of local infiltration anesthesia within the abdomen.

Many descriptions of technique are not clear, or seem very inadequate. These include local anesthesia of the upper jaw, antrum, for glands of the neck, for the tongue, the breast, and for fractures of the extremities; also sacral nerve block, and regional methods for the hand and forearm. For example, the reader is told "If the interior of the nose is to be entered, the nerves supplying this region must be blocked. The local use of anesthetics within the nose does not give good results." He is then left to search for other sections of the text which will describe the nerve supply concerned and the methods of blocking it.

The chapter on spinal anesthesia, written by Arch E. Spelman, M.D., covers the practical aspects of this subject very clearly and well. It deals with the use of procaine only.

Because of the inadequacy of detail of most of the descriptions of technique, this book seems ill suited to the surgeon or anesthetist who wishes either a systematic course of instruction or a handbook for quick reference. It would, however, serve as a moderately helpful adjunct to the definitely superior texts available on regional anesthesia.

Laboratory Medicine: A Guide for Students and Practitioners. By Daniel Nicholson. Second Edition. 566 pp. Philadelphia: Lea & Febiger. $6.50.

The first edition of this work appeared in 1930 and was reviewed in these columns. The present edition is a larger, heavier, and slightly more expensive volume. Many of the interpretations have been expanded and the following procedures have been added: the cough plate for diagnosis of whooping cough, pneumococcus typing by the rapid capsular reaction, Corper's simple method for culturing the tubercle bacillus in sputum, quantitative agglutination tests for the typhoid-paratyphoid group also for undulant fever and tularemia, intradermal tests for echinococcus infection, the patch test in eczema, pregnancy test by Friedman's rabbit method, examination of synovial fluid, urea clearance test for renal function, Congo-red test for amyloid disease, hydrogen-ion concentration of urine, urinary diastase, blood cholesterol reaction, methemoglobin and sulphemoglobin reactions, the histamine test to evaluate circulation of the lower extremities and the use of the ultropak microscope in rapid tissue diagnosis.

The illustrations have been increased from one hundred and eight to one hundred and twenty-four and are as well reproduced as they were in the first edition. For those who wish an excellent reference volume on laboratory medicine Dr. Nicholson's book fulfills all requirements.